The JOURNAL *of the Oklahoma State Medical Association*

Volume 49 **OCTOBER 1956** Number 10

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THE JOURNAL is the official publication of the Oklahoma State Medical Association, and is published monthly except semi-monthly in April, under the direction of the Council, 601 N.W. Expressway, P.O. Box 9696, Shartel Station, Oklahoma City 3, Oklahoma. Publication office (printer): 111 South Peters Ave., Norman, Oklahoma. Send all change of address notices to P.O. Box 9696, Shartel Station, Oklahoma City, Oklahoma. Subscription to **THE JOURNAL** is included in membership fee. Other subscriptions are $5.00 per year or $1.00 per copy with each request subject to approval of the Editorial Board.

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References: (1) Felix, N. S.: *Pediat. Clin. North America* **3**:317, 1956. (2) Joron, G. E.; Fowler, A. F.; de Vries, J.; Reid, G., & Mathews, W. H.: *Canad. M. A. J.* **73**:956, 1955. (3) Weil, A. J., & Stempel, B.: *Antibiotic Med.* **1**:319, 1955. (4) Perry, R. E., Jr.: *North Carolina M. J.* **16**:567, 1955. (5) Jones, C. P.; Carter, B.; Thomas, W. L., & Creadick, R. N.: *Obst. & Gynec.* **5**:365, 1955. (6) Murphy, F. D., & Waisbren, B. A., in Murphy, F. D.: Medical Emergencies: Diagnosis and Treatment, ed. 5, Philadelphia, F. A. Davis Company, 1955, p. 557. (7) Altemeier, W. A.; Culbertson, W. R.; Sherman, R.; Cole, W.; Elstun, W., & Fultz, C. T.: *J.A.M.A.* **157**:305, 1955. (8) Horton, B. F., & Knight, V.: *J. Tennessee M. A.* **48**:367, 1955.

MEDICAMENTA VERA

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Editorials

Joe N. Hamilton

The press of Oklahoma has been lavish in its tributes to Joe Hamilton on the occasion of his retirement as executive secretary of the Oklahoma Society for Cripple Children, a post which he has held since 1925. He richly deserves all of them and much more. He has for over 30 years been Mr. Cripple Children's Society. The designation implies a limitation and there is no limit to the good that is in the man nor has there been a limit to his interest in all phases of human welfare in Oklahoma.

This interest accompanied by a high order of intelligence and understanding has made him the best public relations man Oklahoma medicine has ever had. The work of the Society began when roads were bad, automobiles slow and temperamental, tires were high pressure ones easily punctured by horse shoe nails and when people in small towns and on the farms were often isolated and quite unaware of developments outside their immediate environment. Radios were few, talking pictures were still in the future and the World Series was followed on a blackboard in front of the newspaper office. Many of the men practicing medicine in what had been Indian Territory had had little formal medical education and had been licensed to practice under the grandfather clause.

These were the people that Mr. Hamilton and his teams went among to set up clinics for the evaluation of children who were crippled. Nothing could come of this unless remedial measures could be instituted where indicated. This meant that the medical profession and its worth had to be sold to the parents and very often to grandparents, uncles and aunts. Not infrequently two or three visits a year apart to the clinic would be necessary before the parents could be convinced. The growth and development of the program and the extension of its services to include all forms of crippling is ample proof of a good job of selling.

In his final letter to the members and friends of the Oklahoma Society for Crippled Children, Mr. Hamilton gives some idea of the scope of lay interest in things medical that has been stimulated by him.

"Many official and voluntary organizations and institutions have lent encouragement and contributed materially to the crippled children's program. Special acknowledgments are due the State Department of Education, including particularly the Division of Vocational Rehabilitation; the Oklahoma Commission for Crippled Children; the Oklahoma Hospital for Crippled Children; the State Health Department; the State Department of Public Welfare and the Federal Children's Bureau. Also, much credit is due the Oklahoma Hospital Association, Oklahoma Medical Association, Oklahoma Educational Association, the Tuberculosis Association, Social Welfare Association, National Polio Foundation, Red Cross, the Shriners, the Oklahoma Coaches Association, the various men's civic clubs, and the many women's organizations including Junior Leagues' farm study groups and the Tulsa Cerebral Palsy Association.

"I desire to call special attention to the contributions of churches of Oklahoma and to the Oklahoma press. For 30 years, facilities of the various church plants have been made available without charge for diagnostic crippled children's clinics. The various papers of the State have given liberally of both their editorial and news space to the promotion of crippled children's clinics and of Easter Seal sales. The same words of appreciation can be heartily extended to our broadcasting stations.

"In my final official letter to the membership and other friends of the Oklahoma Society, it would be amiss if I did not pay special tribute to the one man, who for more than thirty years paid me for doing what I should like to have done anyway. The reference, of course, is to the late L. H. Wentz, formerly a fellow citizen and Rotarian at Ponca City ,who in the latter part of 1925, invited me to become executive secretary of the then newly organized So-

ciety for Crippled Children. He agreed to underwrite the project financially. Not only did he do this consistently, but he contributed liberally of his personal time and energies in the development of a state-wide program. During his lifetime, he served as director and treasurer of the Society."

We, of the profession, wish to extend our thanks to Mr. Hamilton not only for the things he has done for the crippled child but also for making the public aware of the fine things medicine has to offer to the crippled and unwell of all ages. No man has done more to blend the knowledge and know-how of the profession and the interest of the lay public into a force for improving physical welfare. Mr. Hamilton is and has been one of the first people of Oklahoma and all who know him regret that he has reached the age of retirement.

Our New Home

As I audited a council meeting yesterday, the first one in the new building, my mind kept wandering from the proceedings to the meaning of it all.

The profession as it has been in Oklahoma is perishable. Time drops us off the vine. It is difficult even for us who are part of it to grasp permanency out of an intangible represented only by the human being. How much more difficult must it be fore a lay person or members of a lay organization to see solidarity and permanency in an organization whose existence is known only by the members known to them.

The new building is a material thing, beautifully designed and located and functionally near perfect. It cost each of you 35 dollars. But you, the administration and the building committee have created a picture in the minds of the doctor and of the public of a permanent structure which is not the building but the medical profession itself. It should go a long way toward driving home to the doctor and to the public the responsibility in perpetuity that the State Association feels toward all matters that have to do with the health of the community which its members serve.

If you have been irked by assessment levied by the House of Delegates pay your new home a visit. If you moan for the young man who has to also pay it, just bring him or send him for a visit too. If you think it is too far from you, think where you could have placed it that it could better serve the purpose for which it is intended. It is on the highway 66 By-pass, which joins the Turner Turnpike, highway 77 By-Pass and highways 74, 3 and 270 on the northwest and is only a short distance north of highway 62 as it passes the State Capitol. One could not have found a location in the capitol city more accessible to members driving in from everywhere in the State.

Open House Planned

Members of the Association will soon be invited to attend an open house and dedicatory ceremony at the new State Headquarters Building. Slow delivery of new furnishings prohibits the establishment of a definite date at this time. Personal invitation will be sent to each member well in advance.

Scientific Articles

FLUID *and* ELECTROLYTE *Problems in Clinical Medicine*

HELEN EASTMAN MARTIN, M.D., and TELFER B. REYNOLDS, M.D.

This article appeared in the Medical Bulletin of the University of Southern California, April, 1956. Because of the quality of the material and the clarity of its presentation, permission was requested to reprint it for the profession in Oklahoma through the Journal. This permission was kindly granted by the editor.

In 1950 a general article on fluid and electrolyte problems[1] was presented in this journal. Since that time there has been considerable expansion of knowledge concerning these problems, both at the basic and clinical level. As there are now several excellent short and long books in this field[2, 3, 4, 5] it is not the purpose of this article to present a short general review of the subject. However, it is our purpose to present briefly two topics of considerable clinical interest (osmolarity and the sodium problem and acid-base relationships) in which there has been enough new knowledge added or re-orientation of knowledge to make discussion worthwhile. The authors have drawn heavily on standard works in the field of fluid and electrolyte metabolism, augmented by their own experience.

Osmolarity, Sodium and Distribution of Body Water

As laboratory measurements of electrolytes in serum are expressed as concentrations—so many milliequivalents per liter—much of the clinical thinking about electrolytes has centered around alterations of concentration of any given electrolyte. Due to difficulties of determination of total body water, or even plasma or blood volume for immediate clinical use, clinicians frequently have not thought about total amounts of electrolyte in the body. The serum concentrations alone do not tell us whether there is a normal, increased, or decreased amount of any electrolyte. Only a clinical estimate of the state of body hydration, plus the estimation of internal distribution of body water, will help us—so that we can multiply volume by concentration to see if there is an increased, normal, or decreased total amount of electrolyte.

In the last analysis the *osmolarity* of the blood governs, via complicated pathways, the retention or rejection of water and hence electrolyte concentrations.

What do we mean by osmolarity?

An effective osmotic pressure is exerted only when there is a membrane (here, the cell membrane) which is permeable to the solvent (H_2O here) but not to all the solutes (here, sodium) and is governed by the number of particles in solution, not by weight or valence.

A few definitions will clarify these differences. A *mol* is the gram molecular weight of a substance. An *osmol* is the gram molecular weight of a substance divided by the number of ions into which it dissociates in a solution. As sugar does not dissociate in solution, one mol — 1 osmol, but a substance such as sodium chloride that almost completely dissociates into sodium and chloride in solution has 2 osmols for every gram molecular weight in solution.

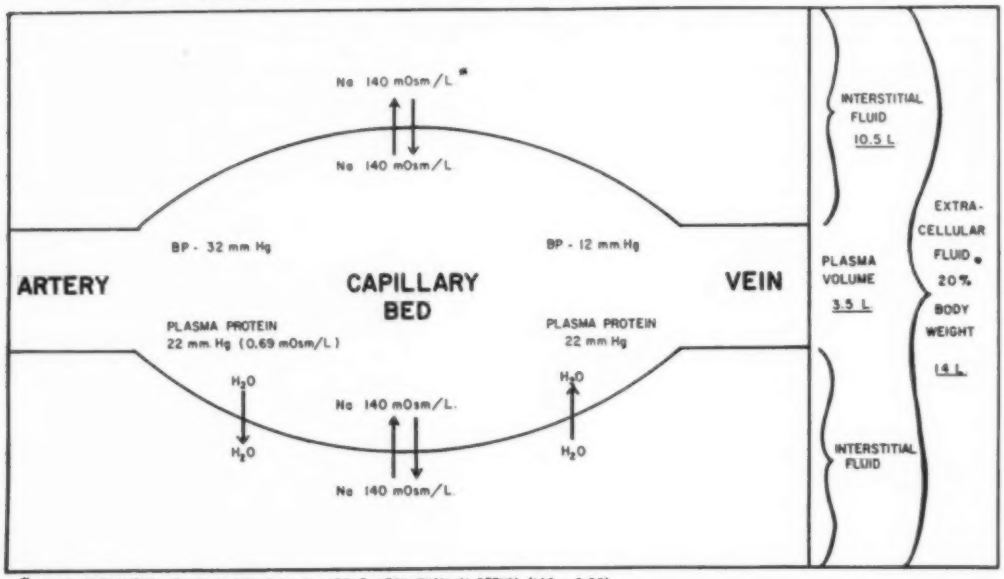
one mol — 2 osmols

$NaCl \quad Na^{+} + Cl^{-}$

A *milliosmol* is 1/1000 of an osmol. A *milliequivalent* is a means of quantitating electrolytes, which ignores their mass and takes into account only their electrical charge. An equivalent weight therefore is defined as a gram-ionic weight if the valence is one, or half a gram-ionic weight if the valence is two, etc. The strength of a solution with regard to an electrolyte is usually expressed as milliequivalents per liter.

DIAGRAM 1

FLUID MOVEMENT BETWEEN BLOOD VESSELS AND INTERSTITIAL FLUID



* Na IN INTERSTITIAL FLUID IS ACTUALLY A LITTLE LESS THAN IN SERUM (140 x 0.95) BECAUSE OF GIBBS-DONNAN EFFECT OF NON-DIFFUSIBLE PROTEIN.

The following illustrations (Tables 1 and 2) will point up these differences between weight (mgms), electrical charge (equivalence), and particles in solution (osmolarity).

I. Sodium and Blood Volume

A study of Diagram 1 shows clearly that the osmolarity of sodium does not govern water movement between the plasma and interstitial fluid as sodium can move freely between these two compartments. The plasma proteins which cannot leave the vascular bed, and thus have an effective osmolarity because of this and the hydrostatic pressure (the blood pressure), govern movement of water out of and into the capillaries according to the pressure gradient in the Starling hypothesis (see Diagram 1).

Indirectly the concentration of sodium does influence plasma and total extracellular volume, for when the serum sodium rises above normal there is hyperosmolarity. This affects the osmolar receptors of the hypothalamic area and causes an increase of antidiuretic hormone and H_2O retention, to restore osmolarity and concentration. Conversely, when there is a low serum sodium there is decrease of antidiuretic hormone and H_2O excretion. Also, as discussed in detail later, changes of osmolarity of the serum govern water movement into and out of cells. Volume becomes more important than concentration when there is threat of loss of extracellular volume—particularly blood volume—as the homeostatic mechanisms of the body are all set with stress and fluid loss to conserve sodium and water to the body. The mammalian kidney represents a long evolutionary development from the saline envir-

TABLE 1

Comparison of Weight, Equivalence and Osmolarity

	Millimols (mgm)	Valence	Milli-equivalents	Milli-osmols
Sodium	23	1	1	1
Calcium	40	2	2	1
Glucose	180	—	—	1
Albumin }	70,000	varies	23	1
Globulin }	160,000	with pH		

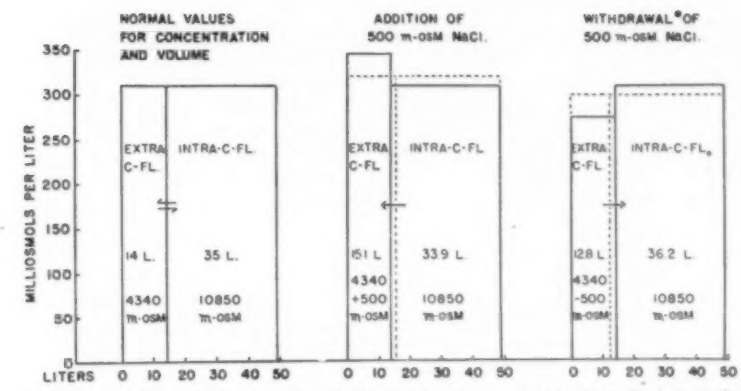
TABLE 2

Comparison of Weight, Equivalence and Osmolarity in Serum

	mg/100 cc	mEq/L	mOsm/L
Sodium	330	143	143.0
Calcium	10	5	2.5
Glucose	100	—	0.55
Protein (albumin globulin)	65,000	16 (at pH of serum)	0.69

DIAGRAM 2

THE DARROW — YANNET DIAGRAM



(Courtesy J. L. Gamble: *Chemical Anatomy, Physiology, and Pathology of Extracellular Fluid*, Cambridge, Harvard University Press[6])

onment of the sea to the sodium deficient environment of the air and earth. The kidney, primed by hormonal influences—activation of sodium-retaining hormones under stress situations—responds by maximal sodium retention.

II. Sodium as the Distributor of Total Body Water

It is assumed that the osmolarity of the extra- and intracellular compartments is the same. As sodium cannot enter the cell, except in small amounts, but water can, the osmolarity of sodium will govern the movement of water*, in order to equalize the osmolarity of the two compartments (see Diagram 2).

Thus, if sodium is added to the extracellular compartment the osmolarity rises higher than in the cells and water moves from the cells until the extracellular and intracellular osmolarity equalizes at a higher than normal figure. To illustrate, if the extracellular osmolarity is increased by the addition of 500 m Osm/L of NaCl (about 15 grams) without water, in order to equalize intra- and extracellular osmolarity water will move out of the cells as per the following calculations:

Total m Osm in body normally

Extracellular

$14L \times 310 \text{ m Osm/L} = 4340 \text{ m Osm}$

Intracellular

$35L \times 310 \text{ m Osm/L} = 10850 \text{ m Osm}$

Total body fluid

$49L \times 310 \text{ m Osm/L} = 15190 \text{ m Osm}$

1. New temporary osmolarity of extracellular compartment

$$\frac{4340 + 500}{14L} = 346 \text{ m Osm/L (increase in osmolarity)}$$

2. New Total osmolarity/L in all body fluid

$$\frac{15190 + 500}{49L} = 320 \text{ m Osm/L}$$

3. Adjusted volume of extracellular compartment

$$\frac{4340 + 500}{320} = 15.1 \text{ Liters}$$

New extracellular volume = 15.1 Liters (gain 1.1 L)

New extracellular osmolarity = 320 m Osm/L

New intercellular volume = 33.9 Liters (loss 1.1 L)

New intracellular osmolarity = 320 m Osm/L

If sodium without water is lost from the extracellular compartment, osmolarity falls and water moves into the cells until the two compartments' osmolarity is similar, but lower than the normal level. To illustrate, if 500 m Osm of NaCl are lost from the ex-

*The movement of water is, of course, determined by the difference between the total osmolarity due to sodium and associated ions in the extra-cellular fluid, and potassium, protein, magnesium, and associated ions within the cell. Shifts in water can be produced by changes in the total amount of osmotically active material inside the cell (due to changes in potassium, magnesium, phosphate, or changes in pH). However, changes in sodium in the extracellular fluid are of primary importance, and other factors are assumed to be unchanged in the present discussion.

TABLE 3

Serum Sodium Levels on Entry in 19 Patients in Diabetic "Coma"

Serum Sodium Level		
Over 145 mEq/L	Normal 135-145 mEq/L	Under 135 mEq/L
11 patients	5 patients	3 patients

tracellular compartment, water will move into the cells as per the following calculations in order to equalize intra- and extracellular osmolarity:

Total m Osm in body normally

Extracellular

$14L \times 310$ m Osm/L $=$ 4340 m Osm

Intracellular

$35L \times 310$ m Osm/L $=$ 10850 m Osm

Total body fluid

$49L \times 310$ m Osm/L $=$ 15190 m Osm

1. New temporary osmolarity of extracellular fluid

$$\frac{4340 - 500}{14} = 274 \text{ m Osm/L}$$ (decrease in osmolarity)

2. New total osmolarity/L in all body fluid

$$\frac{15190 - 500}{49} = 300 \text{ m Osm/L}$$

3. Adjusted volume of extracellular compartment

$$\frac{4340 - 500}{300} = 12.8 \text{ Liters}$$

New extracellular volume — 12.8 Liters (loss 1.2 L)

New extracellular osmolarity — 300 m Osm/L

New intracellular volume — 36.2 L (gain 1.2 L)

New intracellular osmolarity — 300 m Osm/L

III. Clinical Interpretation of Serum Sodium Levels

If the extracellular volume varies and the total body sodium remains the same, one could have low or high serum sodium levels depending on H_2O increase or loss. Conversely, if the extracellular volume remains normal and total body sodium varies, one could have low or high serum sodium levels depending on sodium increase or loss. If both extracellular volume and total body sodium vary one can have normal, low, or high serum sodium levels (see Diagram 3).

Clinical Significance of Serum Sodium Levels

Does the serum sodium level reflect total body sodium?

As indicated above, unless the fluid volumes or total body sodium levels are known, serum sodium levels only tell concentration.

This can be documented by experiences in patients who have lost or gained sodium and water. All would concede that patients in serious diabetic ketosis and acidosis have lost body sodium with the polyuria attendant upon hyperglycemia and glycosuria. Table 3 shows the entry serum sodium levels in 19 patients with diabetic coma. Eleven of the patients or 58 per cent had low levels, 5 of the patients or 26 per cent had normal levels, and 3 of the patients or 16 per cent had high levels, despite the fact body sodium was depleted in all.

Table 4 shows the serum sodium levels in a group of patients—all with deficits of body sodium and water due to gastrointestinal fluid loss. Four of these patients or 21 per cent had high serum sodium levels, with depleted body sodium levels; 9 of these patients or 47 per cent had normal serum sodium levels despite decrease in total body sodium and 6 of these patients or 31 per cent had low serum sodium levels associated with low total body sodium.

Table 5 shows the serum sodium levels in a group of patients with oedema due to a variety of causes, all of whom had obvious increases of total body sodium and water. Ten of these patients or 62½ per cent had a low serum sodium, despite increased total body sodium, and 6 or 37½ per cent had normal serum sodium levels, despite an increase in total body sodium.

TABLE 4

Serum Sodium Levels in 19 Patients with Gastrointestinal Fluid Loss

Diagnosis	mEq/L Serum Sodium		
	High over 145 No. Pts.	Normal 135-145 No. Pts.	Low under 135 No. Pts.
Vomiting due to pyloric obstruction	3	5	1
Vomiting due to other causes	1	1	
Diarrhea		2	2
Intestinal obstruction		1	2
Bile peritonitis			1

TABLE 5

Serum Sodium Levels in Patients with Oedema

Category	mEq/L Serum Sodium	
	Normal 135-145 No. Pts.	Low under 135 No. Pts.
Congestive heart failure	5	7
Cirrhosis with ascites and oedema	1	2
Nephrotic syndrome		1

DIAGRAM 3

Relation of Serum Sodium Concentration to Changes in Extracellular Volume or Total Body Sodium

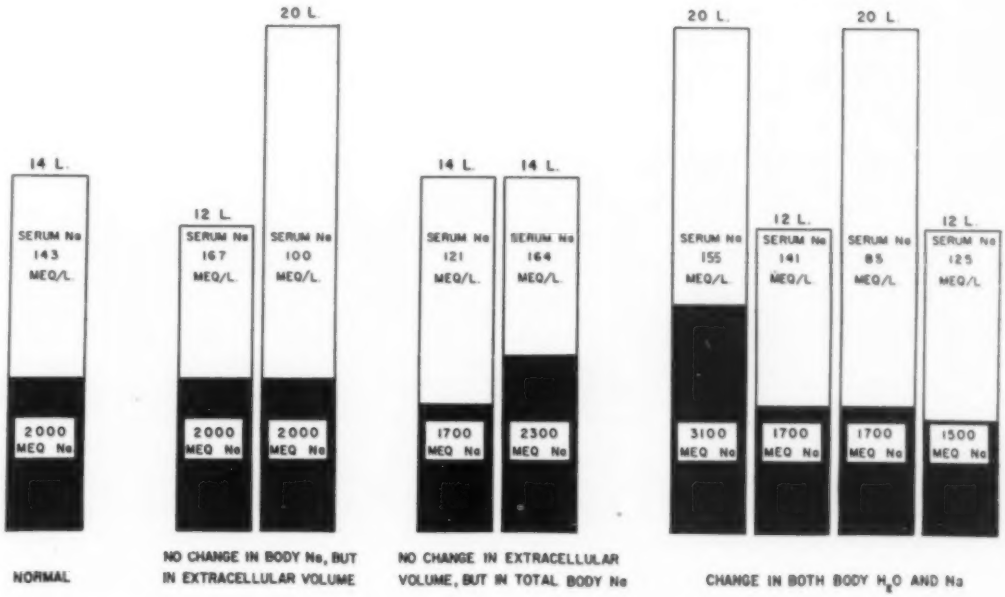


Table 6 plus the history of water and electrolyte gain or loss (vomiting, diarrhea, weight gain, excess sweating, polyuria or oliguria), plus the physical impression of the patient as to presence of oedema, dehydration, or normal hydration, plus the hematocrit, or total protein, and the urea or non protein nitrogen level, should make it possible to interpret most serum sodium levels.

Interpretation of a serum sodium level as to deficit or excess of body sodium, distribution of body water, and treatment of the patient may be considered as follows:

1. Low Serum Sodium

The serum sodium may be low with high, normal or low total body sodium (see also Diagram 3).

Category 1. When the serum sodium is low, plus low hematocrit and total protein, and *there* is *no oedema*, total body sodium is presumably normal and the low serum sodium is due to water retention. This is seen commonly immediately postoperatively—and is thought to be due in part to activation of antidiuretic hormone with oliguria and water retention[4]. The treatment here is not *extra* sodium, but just the usual maintenance amount of sodium plus that lost, and no overloading with water.

Category 2. When the serum sodium is low, the hematocrit and total protein low, and *there is oedema*, total body sodium and water are increased. This situation may be seen in oedematous states, as congestive heart failure, cirrhosis, and nephrosis, where there may be either an isotonic expansion of extracellular fluid or a hypotonic expansion with retention of more water than sodium. The treatment is to eliminate both water and sodium via diuretic therapy—plus some restriction of water. *Hypertonic sodium solutions should not be used* as the body already contains too much sodium and pulmonary oedema may be precipitated by increasing the osmolarity of the extracellular fluid and drawing water out of the cells (see Diagram 2).

Category 3. When the *serum sodium* is low, with increased hematocrit or total pro-

TABLE 6

Interpretation of Serum Sodium Levels

Category	Serum Na Level	Total Body Na Level	Total Body H_2O Level	Urea N or NPN Level	Hematocrit	Total Protein	Type of Change	Clinical Condition	Therapy
1.	Low ↓	Normal	Increased ↑	Normal or low	Low ↓	Low ↓	Hypotonic expansion due to retention of H_2O	Immediate post-operative states	Nothing, except maintenance fluid and fluid lost
2.	Low ↓	Increased ↑	Increased ↑	Normal or low	Low ↓	Low ↓	Hypotonic expansion due to retention both H_2O and Na, but more H_2O than Na	**Oedematous states:** congestive failure, cirrhosis, nephrosis	Diuretic Rx. Na restriction H_2O restriction occasionally
3.	Low ↓	Low ↓	Low ↓	Increased ↑	Increased ↑	Increased ↑	Hypotonic contraction with loss of Na than H_2O	**States of fluid loss** G.I. Fluid: (vomiting, diarrhea, etc.) Polyuria*	Na containing fluids—usually isotonic. Rarely hypertonic
4.	Normal	Increased ↑	Increased ↑	Normal or low	Low ↓	Low ↓	Isotonic expansion	**Oedematous states:** congestive failure, cirrhosis, nephrosis	Diuretic Rx. Na restriction
5.	Normal	Low ↓	Low ↓	Increased ↑	Increased ↑	Increased ↑	Isotonic contraction	**States of fluid loss** G.I. Fluid: (vomiting, diarrhea, etc.) Polyuria*	Isotonic Na containing fluids, type depending on acid-base balance
6.	Increased ↑	Normal	Low ↓	Normal or increased	Increased ↑	Increased ↑	Hypertonic contraction with loss of H_2O	H_2O deficits in patients unable to drink	Hypotonic solutions or glucose in H_2O
7.	Increased ↑	Low ↓	Low ↓	Increased ↑	Increased ↑	Increased ↑	Hypertonic contraction with loss or more H_2O than Na	**States of fluid loss** G.I. fluid (vomiting, diarrhea, etc.) Polyuria*, excess sweat	Isotonic Na containing fluids. Rarely hypotonic
8.	Increased ↑	Increased ↑	Increased ↑	Normal or low	Low ↓	Low ↓	Hypertonic expansion with more Na than H_2O retention	**Oedematous states:** congestive failure, cirrhosis, nephrosis	Diuretics, Na restriction ? Force H_2O

*Polyuria as used here means loss of both sodium and water.

tein, an elevated NPN and there is evidence of dehydration, there is a deficit of body sodium. This state is seen when there has been loss of both water and sodium. There can be an isotonic, hypotonic or hypertonic loss of extracellular fluid. If more sodium than water is lost there will be a hypotonic contraction with low serum sodium. This condition may be seen where there is loss of gastrointestinal fluid of any type or any type of polyuria associated with sodium loss. Treatment consists of the use of isotonic sodium-containing solutions; very rarely a combination of isotonic and hypertonic sodium solutions is required if the sodium deficit is marked.

2. *Normal Serum Sodium*

Category 4. When the serum sodium is normal, with low hematocrit or total protein and *there is oedema*—total body sodium is increased. Therapy here should be the use of diuretics for elimination of sodium and water, and sodium restriction.

Category 5. When the serum sodium is normal, with increased hematocrit, total protein and elevation of NPN or urea, and there is clinical evidence of dehydration, *there is a deficit of body sodium* due to isotonic *contraction of extracellular volume.* This is seen in conditions of dehydration (vomiting, diarrhea, polyuria). The treatment here is use of isotonic sodium-containing fluids—the type depending on the state of acid-base balance and the presence or absence of impaired renal function.

3. *Elevated Serum Sodium*

When the serum sodium is elevated there may be low, normal, or increased body sodium (see Diagram 3).

Category 6. When the serum sodium is increased and the hematocrit and total protein are low and there is oedema, there is an increase of total body sodium. This condition occurs occasionally in congestive failure— where more sodium than water is retained. Treatment consists in diuretics to remove sodium plus adequate water intake.

When the serum sodium is increased and the hematocrit and total protein are elevated, there may be normal body sodium (Category 7) or low body sodium (Category 8).

Category 7. Represents conditions of

water deficit—with little or no sodium loss. This is seen in patients who are unable to drink or obtain water. Here water must be supplied, as glucose in water or in hypotonic solutions.

Category 8. Represents states of fluid loss (vomiting, diarrhea, polyuria) where there has been a loss of both water and sodium, but more water than sodium. Here isotonic and hypotonic solutions should be used.

Thus it is seen that in the two great groups of clinical disorders affecting sodium and water balance

(1) Loss of water and electrolyte and

(2) Retention of water and electrolyte

there may be normal, low or elevated serum sodium levels, depending on whether the gain or loss has been equal for sodium and water, or more water than sodium, or more sodium than water. This means then that the serum level can only be interpreted as to total change in body sodium by assessing the expansion or contraction of body fluid—by physical estimate of normal hydration, dehydration or oedema, and evidence of dilution or contraction of extracellular volume as indicated by hematocrit, total protein and NPN or urea nitrogen. It must be pointed out, however, that the hematocrit and total protein may not be helpful if they have been previously altered by anemia, malnutrition, or blood loss. The NPN or urea nitrogen may be used only if there is no known previous renal disease or in retrospect, as to change with hydration. This means the clinical estimate of hydration, plus the primary cause of fluid imbalance, is often the only means of assessing the significance of serum sodium levels.

IV. Treatment

It is obvious from the previous discussion then that one does not treat the serum sodium level per se, but the patient, and the underlying cause of alteration of body fluid and electrolyte.

The Low Sodium Syndrome. There has been considerable discussion concerning the low sodium syndrome and hyponatremia. Hyponatremia is used incorrectly if it is used to include symptoms. By definition hyponatremia means only a *low serum sodium.* Normal, high or low serum sodium levels may be associated with no symptoms or certain symptoms.

At the present time we do not know whether symptoms which have been defined as due to low sodium are related to levels of body sodium, relation between serum concentrations and total levels, or the serum levels alone. Most of the symptoms of the so called "low sodium syndrome" (weakness, fatigue, muscle cramps, confusion to coma, and hypotension) could be attributed to decreased blood volume, and decreased cardiac output. It is difficult to separate the volume effects from the ion effects, as sodium so largely controls fluid balance. To date no experiments have been done showing the effects of sodium alone.

States of Dehydration are most apt to give symptoms of the low sodium syndrome. Such states are best treated by isotonic sodium-containing fluids, and where indicated, plasma.

States of Overhydration with hyponatremia (particularly congestive heart failure) are very rarely associated with the symptoms of the low sodium syndrome, and *should not be treated with sodium-containing solutions, particularly hypertonic solutions,* as pulmonary oedema may be precipitated.

High serum sodium levels due to water lack indicate need for glucose in water, when total body sodium is normal. If due to loss of extracellular volume, isotonic or hypotonic sodium-containing solutions should be used (see below).

Tonicity of Fluids. Fluids are divided into isotonic, hypotonic and hypertonic—indicating an osmolarity that is equal, less than or more than that of blood plasma.

While glucose in water is isotonic to blood due to the glucose content, it does not reconstitute extracellular volume — since the glucose is used for metabolism and the water runs out in the urine, as it does not remain in the body without sodium, except in unusual circumstances.

Hypertonic Solutions. Due to their increased osmolarity these solutions are used as diuretics—due to their effect in drawing water out of the cells (see Diagram 2), and due to the increased osmolar load presented

to the kidneys against which water cannot be reabsorbed in the kidney tubule. A very definite contraindication to the use of hypertonic solutions is cellular dehydration, as further depletion of cell water may result. Hypertonic saline is rarely indicated in the treatment of sodium deficits unless there is such a large deficit of total body sodium that there would be a tremendous gain in weight if the deficit were made up entirely with isotonic saline.

The Role of Hypotonic Sodium-Containing Solutions is still to be determined, as to whether they should be used where there is both cellular and extracellular dehydration. How much of the extra water will be retained has not been determined. They do provide extra water if there is an increased osmolar load in the urine, as with glycosuria.

Isotonic Sodium-Containing Solutions. In general, if renal blood flow can be restored the kidney can handle the relative retentions of sodium and water. To this end, in most situations of sodium deficit, isotonic solutions of sodium are usually adequate.

Calculation of Sodium Deficits. It is obvious from the above considerations that it is impossible to calculate the body sodium deficit from serum levels alone—as the serum level may be high, normal or low.

If one desires to calculate the sodium required to correct a low serum sodium level the deficit per liter of sodium must be multiplied by an assumed total body water (42 liters) rather than by extracellular volume (14 liters), since sodium governs total movement of body water due to its osmolar effect.

Example:

Serum sodium level	125 mEq/L
Normal serum sodium level	135 mEq/L
Deficit	10 mEq/L

10 mEq/L × total body water (42 L) =
420 mEq Na = 9.7 gm Na = 24 gm NaCl

However, one must replace the amounts of *sodium calculated cautiously,* and *give ⅓ or* ½ the *calculated* amount originally — then check the patient and the chemistry. This is due to the fact that movement of water which may be primarily responsible for a low serum sodium is affected by sodium administration, and water diuresis may occur. More basically the inability to use calculations is due to the fact that normal homeostatic mechanisms are altered when concentrations are disturbed, and *the serum* level *alone,* as indicated throughout this paper, can lead one astray as to deficits or excesses, or their extent.

ACID-BASE BALANCE

The proton theory of acids and bases developed by Bronsted has been found to provide a better explanation for observed phenomena in the body that the older theory of Arrhenius and has rendered the older terminology obsolete. Proper attention to semantics and knowledge of a few simple chemical concepts greatly facilitate understanding of acid-base balance in the body.

Definitions and Chemical Concepts

Acid

An acid is defined as a compound which when placed in solution dissociates to provide free hydrogen ions or protons (fig. 1). The greater the tendency to form free hydrogen ions the stronger the acid. As examples, hydrochloric acid (HCl) is a strong acid which freely dissociates into hydrogen (H^+) and chloride (Cl^-) ions (fig. 2). Ammonium (NH_4^+) is a weak acid dissociating only slightly into hydrogen ions (H^+) and ammonia (NH_3) (fig. 3).

The acidity of a solution refers to the amount of hydrogen ion (H^+) free in the solution and is often expressed as the reciprocal of the logarithm of the hydrogen ion concentration (pH).

There is a tendency in the older literature to refer to all negatively charged ions or anions (such as Cl^-, SO_4^{2-}, etc.) as acids. This is chemically incorrect.

Base

A base is defined as a substance which has a tendency to combine with hydrogen ions removing them from solution. The strength of this tendency measures the "strength" of the base. Examples are hydroxyl ion (OH^-) or ammonia (NH_3) as strong bases and chloride ion (Cl^-) as a very weak base. By this terminology, then, an acid will always consist of hydrogen ion combined with a base.

Sodium (Na^+) and potassium (K^+), commonly called bases in the older terminology, are best called cations.

Law of Mass Action and Reversible Reactions

At a constant temperature the rate of a reaction involving gases or ions is proportional to the concentration of each kind of molecule or ion taking part in the reaction.

i.e.: $H^+ + HCO_3^- \rightarrow H_2CO_3$

Doubling the concentration of H^+ will double the number of collisions between H^+ and HCO_3^- and so double the rate of formation of H_2CO_3.

For reactions that are reversible the principle of dynamic equilibrium applies.

i.e.: $H^+ + HCO_3^- \rightarrow H_2CO_3$

and $H_2CO_3 \rightarrow H^+ + HCO_3^-$

Such a reaction comes to equilibrium when the rate of formation of H_2CO_3 and its rate of breakdown are equal.

Then $H_2CO_3 \leftrightarrows H^+ + HCO_3^-$

At equilibrium the product of the concentrations of H^+ and HCO_3^- divided by the concentration of H_2CO_3 is a constant value for a given temperature and is spoken of as the dissociation constant for the compound in question. Such a dynamic equilibrium, if any stress is placed upon the system, will adjust itself to maintain equilibrium by reacting in the direction that relieves the stress (Le Chatelier law).

For example: $H_2CO_3 \leftrightarrows H^+ + HCO_3^-$

If H^+ is added to the equilibrium mixture the reaction will shift to the left. The rate of formation of H_2CO_3 will be increased until a new balance is achieved. In the final equilibrium mixture there will be more H_2CO_3 and less HCO_3^- than in the beginning and less H^+ than was added. There would be a similar shift to the left if HCO_3^- were added. If H_2CO_3 were removed from the equilibrium mixture a shift to the left would also occur resulting in a drop in H^+ and HCO_3^- concentrations and replacement of some of the removed H_2CO_3.

Figure 1

A shift in the reaction to the right would result from the addition of H_2CO_3 or the removal of H^+ or HCO_3^-.

As an example the ionization of H_2CO_3 is depicted in Figure 4. At equilibrium a moderate acidity exists resulting from the innate tendency of H_2CO_3 to dissociate. The addition of a large surplus of $NaHCO_3$ to the solution, by forcing the reversible reaction to the left, would greatly decrease the concentration of H^+ in the mixture and increase the amount of H_2CO_3 (fig. 5).

Acids and Bases in the Body

Since our body fluid is a water solution there are of necessity both hydrogen and hydroxyl ions present it it. The quantity of free hydrogen ion in water is very small and stable, however, and there would be no problem were it not for the necessity of transporting considerable amounts of relatively strong acidic substances through the body. Chief among these is carbonic acid (H_2CO_3), formed by the interaction of CO_2 produced in cell metabolism, and H_2O. ($H_2O + CO_2 \rightarrow H_2CO_3$.) Other and stronger acids are H_2SO_4 and H_3PO_4 which result from the oxidative metabolism of phosphorus and sulfur-containing proteins. Any of these acids if loosed in a water solution in even small amounts would dissociate sufficiently to produce free hydrogen ion concentrations many times greater than .00000004 gms per liter (the concentration of H^+ in body fluids).

Since marked variations in H^+ concentration are incompatible with proper function-

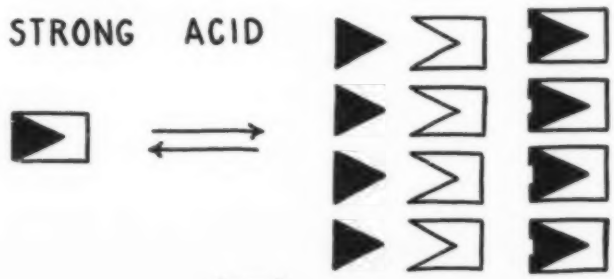


Figure 2

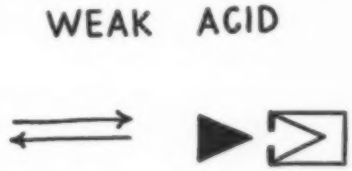


Figure 3

ing of many enzyme systems and other cellular processes, a protective mechanism has evolved in our bodies to minimize potential shifts in H^+ concentrations. There are in the body large amounts of moderately strong bases: HCO_3^-, Hb^- (ionized hemoglobin), and Pr^- (ionized proteinate), and smaller amounts of PO_4^{3-} and HPO_4^{2-}. The tendency of the acids to dissociate into free hydrogen ions is counterbalanced, according to the laws of mass action, by the tendency of the surplus of bases to combine with the free hydrogen ions. All of the common acids in the body have their ionization into H^+ depressed by the presence of large amounts of their constituent bases.

The final equilibrium mixture is such as to satisfy all the mass action equations involving H^+, base and acid. The H^+ ion is in equilibrium with all of the acids and bases since it can presumably pass cell membranes freely even though some of the bases may be fixed within or without the cells (fig. 6).

Remember that wherever there is a negatively charged ion there must of necessity be a positively charged one. The cation partners of these bases are largely Na^+ (for those in the extracellular space: HCO_3^-, PO_4^{3-}, HPO_4^{2-}) and K^+ (for those in the intracellular space: Pr^-, HPO_4^{2-}, and Hb^-).

This sort of a mixture is called a "buffered" one since it strongly resists changes in hydrogen ion concentration. Large amounts of hydrogen ion can be added without marked increase in acidity because of the large surplus of bases available. Of course as the supply of bases is decreased and the amount of unionized acid is increased some drop in pH will occur. The addition of strong base to the mixture will have the effect of removing H^+ by combination with the base. As H^+ concentration drops, however, all of the unionized acids

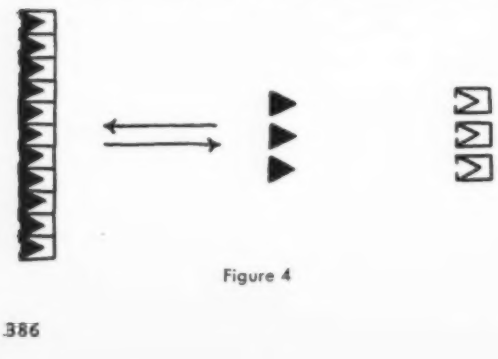


Figure 4

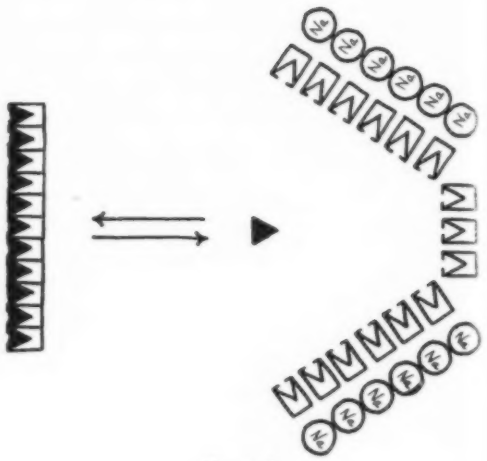


Figure 5

will release H^+ and the alkalinizing effect of the added base will be minimized.

Even though it is a well buffered solution our body fluid could not stay long at the same pH without added protective mechanisms. The constant addition of H_2CO_3, H_2SO_4, and H_3PO_4 from metabolism in addition to organic acids that we can not adequately measure would soon lead to a depletion of bases, a surplus of acids and increased hydrogen ion concentration. This accumulation of acids is prevented by their elimination via the lungs or kidney after being transported through body fluids.

Acid-Base Function of the Lungs

H_2CO_3 is continually eliminated via respiration at approximately the same rate that it is produced in the tissues. The amount of H_2CO_3 in the blood is a function of the ventilatory rate. When for any reason hydrogen ion concentration in the body increases, the respiratory center is stimulated and the carbonic acid blood level is decreased. If, at any time, the blood hydrogen ion concentration falls below normal, less H_2CO_3 is eliminated and its level in the blood rapidly increases. Thus, except under the most extreme circumstances, the lungs can keep the blood pH within reasonably normal limits simply by varying the elimination of carbonic acid. The extreme circumstances would be accumulation, to a marked degree, of other acids than H_2CO_3 or other bases than HCO_3^-.

Acid-Base Function of the Kidneys

Just as the lungs serve to excrete one important metabolic acid (H_2CO_3), so the kidneys excrete the other metabolic acids (H_2SO_4, H_3PO_4, organic acids). In the normal person these substances are formed in amounts of from 20 to 100 mEq/day depending upon the constituents of the diet. A diet high in phosphorus- and sulfur-containing proteins ("acid-ash" diet) may result in the formation of up to 200 mEq of acid/day. If any degree of ketosis were present much larger amounts of keto-acids might be formed. All of these acids ionize to a large degree: their hydrogen ions, therefore, are carried in the body in union with bases while their anions are paired with sodium or potassium.

Example

Let HX be the strong acid resulting from metabolism, ionizing into H^+ and X^-. Let B^- represent an ion of body base (HCO_3^-, HPO_4^{2-}, Hb^- or Pr^-) in combination with Na^+ or K^+. Then $H^+X^- + Na^+B^- \rightarrow HB$ (unionized) $+ Na^+X^-$. If this reaction continued day after day all body bases would soon be combined with hydrogen ion and a progressive acidosis would ensure. In addition the anions of the metabolic acids are largely waste products and must be eliminated.

The renal tubules contain an intricate mechanism whereby both H^+ and X^- may be eliminated in the urine and Na^+ and B^- may be restored to the body. Current concept (unproven) as to how this takes place is as follows: (1) a high local concentration of H_2CO_3 is made possible in the cells of the distal renal tubules because of an unusual concentration of the enzyme carbonic anhydrase. The H_2CO_3 ionizes to some extent into H^+ and HCO_3^- ions. (2) Na^+X^- is present in the tubular urine, having been filtered at the glomerulus. (3) H^+ from the tubule cell is exchanged for Na^+ from the tubular urine. (4) H^+ X^- goes out in the urine and Na^+ HCO_3^- is transferred to the blood stream. (5) $Na^+HCO_3^-$ reacts with HB to yield Na^+ B^- and H_2CO_3. (6) H_2CO_3 is removed by respiration. Thus the original quantity of Na^+B^- is restored and H^+X^- is eliminated in the urine.

The distal renal tubular cell also has the power to form ammonia (NH_3) from the degradation of glutamine (and other amino acids) via the enzyme glutaminase. As H^+ concentration rises in the urine, NH_3 passes from the tubular cell into the urine, and joins with the H^+ to form NH_4^+ ($NH_3 + H^+ \rightarrow NH_4^+$). The NH_3 production keeps the hydrogen ion concentration of the urine from rising to high levels (pH rarely drops below 5.0).

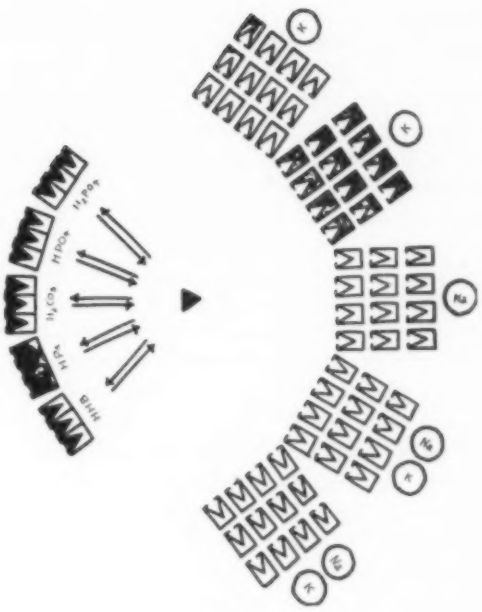


Figure 6

The total quantity of metabolic acid that is handled by the kidney each day is reflected by the total number of hydrogen ions that appear in the urine over and above the number in the glomerular filtrate. The hydrogen ions in combination with NH_3 we can measure as ammonium (NH_4) and the remainder are estimated by titrating the urine back to pH 7.4 (the pH of the glomerular filtrate).

These mechanisms are spoken of as the base-conserving function of the kidney, for obvious reasons. In the normal kidney approximately 1000 mEq. of hydrogen ion can be eliminated (and base manufactured) in 24 hours when the mechanisms are functioning maximally. Several days are required before NH_3 formation reaches its peak, presumably because of a build-up in glutaminase activity.

In addition to their function of disposing of metabolic acids the kidneys, under other circumstances, may have to dispose of ex-

cess base. The reabsorption of $NaHCO_3$ from glomerular filtrate is ordinarily nearly complete. However, if excess $NaHCO_3$ is added to the body (as by ingestion in the treatment of peptic ulcer), then tubular reabsorption becomes incomplete and as much as 1000 mEq. (or more) of $NaHCO_3$ may be eliminated in the urine per day.

The factors that determine tubular HCO_3^- reabsorption are only partially understood—the chief determinant appears to be the pH of the blood. Bicarbonate reabsorption is increased with a fall in blood pH and decreased with a rise in pH.

Clinical Upsets in Acid-Base Balance

In thinking about acid-base problems it is often helpful to construct the equilibrium reaction for H_2CO_3, H^+ and HCO_3^- (see fig. 4). This buffer system represents only 20-30 per cent of the total of such systems in the body but since all of them are in continuous dynamic equilibrium the state of acid-base balance can be accurately assessed by measuring the components of any one of the systems. None of the other buffer systems is susceptible to measurement since they exist in unionized non-reactive form as well as in their electrolyte state.

H^+ concentration can be measured directly with a pH meter. The sum of H_2CO_3 and HCO_3^- can be measured in a Van Slyke apparatus which traps and measures all the CO_2 released when the plasma is acidified (normal 27-29 mEq/L). HCO_3^- alone can be measured by a titration technique (normal 26-28 mEq/L).

I. Acidosis

Acidosis is defined as a drop in blood pH (excess of hydrogen ions). The amount of the change in pH is immediately important since it is presumably this disturbance that interferes with cellular function. The lower pH limit compatible with life is probably approximately 6.8. However, the extent of the loss or inactivation of body base is usually of more importance to us since it provides information as to the duration, likely future course and proper therapy of the upset. Causes of acidosis are as follows:

A. Addition of Excess Acid to the Body

Examples are (1) diabetic acidosis (large amounts of ketoacids from incomplete fat metabolism), (2) ammonium chloride overdose (metabolized to hydrochloric acid), (3) methyl alcohol poisoning (metabolized to formic acid), (4) late stages of salicylate poisoning (acids of undetermined type are formed in excess).

The carbonic acid equilibrium reaction will be displaced to the left by the addition of large amounts of hydrogen ion. HCO_3^- concentration will fall and H_2CO_3 will increase. Much of the added H^+ will be converted to H_2CO_3 by the shift in equilibrium but the final H^+ concentration will still be abnormally high. Ventilation will increase to dispose of H_2CO_3 (to the point of Kussmaul respirations), and the kidneys will excrete maximal amounts of H^+ and NH_4^+ with re-formation of increased amounts of HCO_3^-.

Measurements will disclose a low blood pH and a low HCO_3^- content. If there is a chemical test for the anion of the offending acid it will be positive (i.e., aceto-acetate in the blood and urine, the ferric chloride test in the urine for salicylate, an elevated blood chloride level with ammonium chloride and positive methyl alcohol test in the blood during the first 24 hours after ingestion).

Treatment will be directed first at stopping the original process if possible (i.e., insulin). Next, adequate renal function should be insured (blood, plasma or extracellular fluid replacement) since shock and decreased renal blood flow interfere with the kidney's very important role in correcting acidosis. Third: extra base should be provided as bicarbonate or lactate solution.

B. Loss of Body Base

This is nearly always the result of loss of large amounts of intestinal secretions (including bile and pancreatic juices) which contain relatively large amounts of HCO_3^-.

The removal of HCO_3^- displaces the carbonic acid equilibrium reaction to the right with a drop in H_2CO_3 and a rise in H^+ concentrations. Ventilation increases, further lowering H_2CO_3 to minimize the increase in H^+. Unfortunately the kidney's natural tendency to form increased HCO_3^- is seriously impaired because of the low extracellular volume and resultant drop in renal blood flow that always accompanies the loss of large volumes of gastrointestinal fluid.

Treatment is directed both at restoring extracellular volume (and therefore renal function) and at repairing the HCO_3^- deficit. These objectives can be accomplished simultaneously by giving sodium bicarbonate or sodium lactate solution (lactate is metabolized to bicarbonate).

C. Failure of the Lungs

The lungs fail in their acid-base function when serious emphysema or respiratory muscle failure interferes with the ventilation of CO_2. The partial pressure of CO_2 in the alveoli rises and the blood carbonic acid level increases. The carbonic acid equilibrium is shifted to the right, increasing HCO_3^- and H^+ levels. Much of the H^+ is taken up by the other body bases. The body has no further defense but to increase H^+ excretion in the urine, thereby forming more HCO_3^-. The rise in HCO_3^- level counteracts the increased ionization of H_2CO_3 and returns blood pH towards normal.

Treatment of this disturbance is directed at improving CO_2 ventilation via positive pressure devices or by eliminating bronchial infection or constriction. The danger of oxygen administration without concomitant measures to insure CO_2 ventilation is well known. If CO_2 ventilation decreases abruptly, blood H_2CO_3 may increase to poisonous levels with a marked drop in pH.

D. Failure of the Kidneys

There are two types of renal failure that may upset acid-base balance.

1. Tubular acidosis. The ability of the distal renal tubular cells to transfer H^+ and NH_4^+ into urine in exchange for Na^+ with which to re-form $NaHCO_3$ is impaired. The result is a chronic deficiency of HCO_3^- in the body, increased ionization of H_2CO_3 and acidosis. The urine contains less H^+ and NH_4^+ than normal. There is usually no glomerular disease and none of the features of uremia. Treatment is simply to provide a small supplement of sodium bicarbonate daily (50-100 m Eq) which approximates the amount manufactured each day by the tubules.

2. Inadequate glomerular filtration with azotemia (acute or chronic renal disease or prerenal azotemia).

The anions of the metabolic acids (PO_4^{3-}, SO_4^{2-}, and others that are unmeasured) must reach the tubular urine before the tubular cell transport mechanism that acidifies the urine and re-forms bicarbonate can operate. In uremia these anions are filtered in decreased amounts and accumulate in the body. To the extent that they accumulate, HCO_3^- re-formation lags behind and thus there is a deficiency in the body of as many mEq of HCO_3^- as there is an excess of metabolic acid anions. The deficiency of HCO_3^- results in increased H_2CO_3 ionization and acidosis. Ventilation is increased.

Treatment is directed along three lines: (a) Every effort is made to improve glomerular filtration to a maximum by assuring normal extracellular fluid volume and maximum cardiac efficiency. (b) Protein metabolism is decreased to a minimum to cut down the formation of metabolic acids. (c) The addition of sodium bicarbonate or sodium lactate will decrease the acidosis but may increase extracellular fluid volume to a dangerous level.

II. Alkalosis

Alkalosis is defined as a rise in blood pH (deficiency of hydrogen ions). Again, although the actual pH of the blood is important, it is essential that we know the level of HCO_3^- for proper interpretation and treatment of the disorder. Causes of alkalosis are as follows:

A. The Addition of Excess Base to the Body

This is the simplest but not the most common cause. Ingestion of very large amounts of $NaHCO_3$ may exceed the kidney's capacity for excretion, particularly if there is a disease such as heart failure with a tendency to sodium retention. HCO_3^- levels rise, shifting the carbonic acid equilibrium to the left and decreasing H^+ concentration. Ventilation is decreased and H_2CO_3 levels rise further above normal. The treatment is cessation of $NaHCO_3$ ingestion.

B. Loss of Stomach Acid

Continuous gastric suctioning or obstruction of the pylorus from peptic ulcer may result in losses of large amounts of HCl. Removal of H^+ from the body shifts the H_2CO_3 equilibrium reaction to the right with re-

sultant increase in HCO_3^- and drop in H_2CO_3. Ventilation decreases and H_2CO_3 is retained. In the earlier phases of this disorder H^+ secretion and HCO_3^- formation by the kidney cease and HCO_3^- is actually lost in the urine through decreased tubular reabsorption. Alkalosis does not progress under these conditions. However, when loss of extracellular fluid volume and other electrolytes becomes serious, as it inevitably does when all of the gastric juice is lost, renal circulation and renal function rapidly deterioriate. The urine becomes acid (possibly associated with K^+ deficiency, see below) and HCO_3^- elimination ceases. Systemic alkalosis rapidly progresses under these conditions.

The treatment is directed first at restoring extracellular fluid volume so that renal function can be returned to normal. This can be done only with blood, plasma, or sodium-containing solutions. Logically one would use NaCl rather than $NaHCO_3$ or Na lactate. This sort of therapy will often cure the alkalosis since restoration of kidney function enables excretion of large amounts of bicarbonate.

Additional therapy with hydrochloric acid parenterally will relieve the alkalosis rapidly by shifting the equilibrium reaction back towards H_2CO_3. In place of HCl we customarily use NH_4Cl solution which is metabolized in the liver to urea and HCl. This type of therapy, though it will relieve the acidosis, will not improve the accompanying dehydration and imparied renal function.

Potassium deficiency regularly accompanies this syndrome (due to loss in gastric contents) and since there is much evidence to indicate that it is responsible for part of the poor renal function (paradoxically acid urine) it should be administered early in treatment.

C. Hyperventilation

Excessive ventilation may result from emotional upset, head injury, a mechanical respirator or high altitudes. The alveolar partial pressure of CO_2 in decreased and the blood H_2CO_3 level drops. The equilibrium reaction is shifted to the left with drop in H^+ and HCO_3^- levels. H^+ drop, of course, is minimized by the release of H^+ from the other bases. Tetany may result from the H^+ drop. Renal tubular HCO_3 reabsorption is decreased and HCO_3^- appears in the urine, further decreasing blood HCO_3^- levels which has the effect of stabilizing the fall in H^+.

Treatment depends upon the cause. Tetany can be immediately relieved by CO_2 inhalation or rebreathing in a closed system in which the CO_2 accumulates. Opiates are sometimes helpful in slowing down the rapid respirations.

D. Specific Deficiency of Potassium

In hyperadrenalcorticism or in conditions where K^+ is the only electrolyte lost (usually gastrointestinal problems) a peculiar type of alkalosis sometimes results. The urine is often paradoxically acid and therapy with NH_4Cl does not produce the expected improvement. On the other hand K^+ therapy results in a disappearance of the alkalosis. It is postulated by some that there is a disturbance in the normal intra-extracellular relationships such that H^+ moves intracellularly causing an alkalosis in the extracellular fluid and an acidosis in the intracellular fluid. Supposedly an intracellular K^+ deficiency is the genesis of this disturbance.

This is undoubtedly an oversimplification of a complicated relationship but serves to point out the importance of K^+ balance in acid-base problems.

Conclusion

In the analysis of an acid-base problem we want to know (a) the concentration of hydrogen ion in the blood, and (b) the amount of body base, which we infer from the bicarbonate concentration. We are concerned with the function of the lung and the kidneys since serious acid-base disturbances almost never occur unless these organs are temporarily or permanently deranged. Our treatment is first directed at restoring pulmonary or renal function and next at adding hydrogen ion (in the form of ammonium chloride) or base (in the form of sodium lactate or bicarbonate) if they are needed.

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A Survey and Report of An Unrecognized Case:

APPENDICITIS *and* PERITONITIS *During* PREGNANCY

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Appendicitis during pregnancy merits considerable concern because pregnancy, or its commoner complications, tend to obsure prompt diagnosis and because physicians have a pronounced unwillingness to perform surgical operations on pregnant women. Should abortion or miscarriage result after an operation which could have been avoided, or which was done in good conscience for an erroneous diagnosis, the physician may reproach himself unduly. However, the results of delayed surgery can be tragic for both mother and fetus.

The most radical attitude noted in the literature is the advice that any woman who has ever had an attack of appendicitis should undergo an interval appendectomy before she is "permitted" to become pregnant.

Statistics relating to appendicitis during pregnancy vary widely. Laforet[3] quotes an incidence of 1.02 per cent in a survey of the world literature since 1911. The lowest incidence, 0.03 per cent, is quoted by Smith and Bartlett[7]. Most authors call attention to the fact that upward displacement of the cecum by the enlarging uterus and loss of abdominal muscle tone during pregnancy tend to distort and obscure the usual signs by which appendicitis is recognized. Incidence of rigidity of abdominal muscles varies from 10 per cent[4] to 30 per cent[5].

Incidence of gangrene and perforation is infrequently described in the literature, but Smith and Kendall[6, 7] report 61 acute surgical emergencies of the abdomen during pregnancy, 24 (39 per cent) of which were appendicitis. Of these, 4 (16.6 per cent) of the appendices were perforated. Three (75 per cent) of the patients with perforating appendicitis died. Laforet[3] quotes 30 per cent maternal mortality when peritonitis was present.

In the Memorial Hospital at Lawton, in which 90-95 per cent of the obstetric service consists of private patients, 2,884 patients were admitted for delivery on the Obstetric Service in the first four and one-half years of operation. During this time, five appendectomies were done during pregnancy. Since four of the diagnoses were verified pathologically, the corrected incidence is 0.138 per cent. (These statistics may be somewhat inconclusive because a certain proportion of a large number of military dependents residing here get part of their prenatal care from local physicians, then, after the sixteenth week, or in an emergency, become eligible for care at a nearby Army Hospital and transfer there.) Only one of our cases was perforated and obscured diagnosis makes it worth adding to the literature for statistical value.

Case Report

M.B., 25 year old Negro para iv, gravida v, had been seen during the latter half of pregnancy. She could not estimate when her last menses had occurred. Her physician, by observing the rate of uterine enlargement, had estimated the pregnancy to be at term about the end of March, 1956. In January, 1956. she was treated with sulfisoxazole by an urologist for a resistant urinary tract infection. When she consulted her usual physician in February she complained of generalized abdominal pain, vomiting, dysuria and urinary frequency. A catherized specimen contained innumerable pus cells and 10-20 red blood cells per high-power

field. On February 5, 1956, she was hospitalized because of persistent cystitis and for institution of fluid replacement therepy because of dehydration. On February 8, 1956, she developed definite labor contractions. Her temperature was 99.8 (F), RBC 4.43, Hgb 12.75 gm, WBC 16300 with 79 per cent segmented polymorphonuclears and 4 per cent non-segmented. BP was 110/60. Because labor failed to progress, a flat roentgenogram of the abdomen was obtained. It revealed unsuspected twins, the presenting one lying transversely. Auscultation by the attendant failed to locate fetal heart tones and consultation was requested. The consultant concurred in the diagnosis of twin pregnancy with abnormal presentation; premature nonprogressive labor; and persistent infection of the urinary tract. The fetal heart tones of the presenting twin were located and estimated to be about 180 beats a minute. No heart tones of the upper twin were located. The cervix was rigid and not dilated after 14 hours of labor. Toxicity from the infected urinary tract was thought to account for premature labor and fetal anoxia. Caesarian delivery was deemed to be safer for the already anoxic babies than manipulative delivery from below.

With the patient under nitrous oxide-cyclopropane anesthesia a low cervical cesarean section was done. The parietal peritoneum and anterior surface of the uterus had no unusual appearance. The bowel was not inspected when packed away from the sides of the uterus. The bladder flap elevated easily. Small, viable twins, male and female, were delivered through a longitudinal incision in the lower uterine segment. During closure of the uterus, the odor of colon bacillus was transiently perceived twice. Brief consideration was given to the possibility of peritonitis due to a perforated viscus, but the furtive passage of flatus by an assistant was deemed more probably the source. After replacement of the bladder flap, the packs were removed and a literal flood of pus observed on the right side revealed the source of the odor. Suction was instituted and the pus was found to extend as high as the subphrenic space. No activity of the omentum or reaction of immunity in the peritoneum was noted. A completely disintegrated appendix was found attached by a shred to the cecum and extending medially behind the uterus. It was ligated and removed. Penrose drains were inserted through a stab wound in the right flank to the parocolic gutter, the pelvis and the area medial to the cecum, and the laparotomy incision closed. The patient was given 500 cc. whole blood during the operation and another 500 cc. 10 hours later. Shock prophylaxis, duodenal suction, antibiotics and electrolyte maintenance were used postoperatively. One twin, weighing 3 pounds 10 ounces, died on the second day. The other, weighing 3 pounds 1 ounce, died on the fifth day. The mother's convalescence was stormy and continued to be complicated by persistent pyuria. Bateriologic study for antibiotic sensitivity yielded a negative culture.

The drains were removed on the third and fourth postoperative days. An anticipated fecal fistula failed to develop and the patient's clinical course became more routine until the fourteenth day when a subfascial abscess in the lower portion of the incision was noted. It drained spontaneously before surgical drainage could be scheduled, and she became afebrile. She was discharged March 3, 1956, the twenty-third postoperative day.

Comment

This case, like that of Hoffman and Suzuki[2], was completely unrecognized and, like that of Ziegler and Lawrence[9], was discovered in the course of doing a low cervical caesarian section. Like the latter authors, I do not advocate the handling of this case as the method of choice; I report the case only for its unusual features and statistical value.

Summary

A brief review of recent literature surveying the incidence of appendicitis during pregnancy has been presented. A case of unrecognized perforating appendicitis with general peritonitis, obscured by persistent infection of the urinary tract and discovered during a caesarian section for fetal emergency, has been presented for its statistical value.

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The USE *of* CONTACT LENSES

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Corneal pathology is responsible for approximately 14 per cent of blindness in the United States. In our total blind population of some 400,000 this means at least 56,000 are totally blind from corneal pathology and many more with serious visual impairment. Blindness is a problem which concerns us all, not only from its humanitarian aspect but as a major economic problem. Blindness has no significant effect upon mortality, and blind people live their full span of years, often totally or at least partially dependent on others. Improved methods of dealing with corneal abnormalities are thus not only of importance to the opthalmologist, but to all of us by enabling us to give our patients sound advice which has not been culled from the popular magazines.

Many of these patients have congenital defects such as keratoconus and associated conditions which can be successfully treated but for which no means of rehabilitation were available until the past few years. Keratoconus is a developmental anomaly with a definite hereditary tendency involving a weakness of the corneal tissue with stretching in the axial part producing a distortion which is not correctable with conventional glasses. It is usually bilateral, becomes manifest in early adolescence and progresses to scarring with loss of useful vision. Attempts have been made to correct the optical defect with contact lenses for the past 125 years but it is only within the past few years that practical lenses have become available. Early lenses were successful in rare cases only. With the advent of local anesthesia more accurate fitting became possible.

In 1936, when the Zeiss lenses made in Germany were no longer available, Mr. Obrig and others in New York initiated the manufacture of contact lenses in this country. Lenses were made quite accurately from molds obtained from plastic impressions of the cornea and refractive corrections were incorporated in the lenses. However, even these lenses were seldom tolerated for more than a few hours since it was necessary to use a solution in the lenses which was held in by the scleral portion of the lens forming a closed system. This interfered with the normal respiratory function of the cornea and so disturbed the metabolism that corneal edema quickly developed with blurred vision, painful, red and watery eyes. In 1948, Dr. Touhy of Los Angeles developed a fluidless lens which covered only the corneal portion of the eye. This corneal lens allows a circulation of lacrimal fluid behind the lens and produces very little disturbance in metabolism other than the initial symptoms produced by the presence of a foreign body in the eye. Although it is not recommended, patients have kept these lenses in their eyes continuously over a period of weeks with no discomfort or evidence of ocular injury. At first it was thought that this corneal lens could not be used for the correction of keratoconus because of its small size so fluidless lenses of a corneo-scleral type were developed with perforations or other devices which allowed circulation behind the lens.

In my experience the corneal lens has satisfactorily corrected every case of keratoconus that could be corrected with the corneoscleral lens slides. Those cases which were too far advanced to be corrected with any type of contact lens must be treated by partial penetrating keratoplasty. In 21 cases of keratoconus, seen in the past five years, only four have required surgical treatment and these obtained perfect results, confirming the experience of other surgeons that corneal transplantation in keratoconus has a good prognosis.

In addition to their use in keratoconus, contact lenses are indispensable in many other types of corneal pathology. Corneal injures frequently leave scars which prevent useful vision. In some of these patients the contact lens forms a new corneal surface which relieves the distortion sufficiently to allow useful vision. One such patient in our series was completely disabled with bilateral chemical burns that produced complete blindness in one eye and reduced vision in the other eye to perception of hand movements. By some miracle of optics he is able to get 20/20 vision with one eye with a contact lens, has useful reading vision and is once more gainfully employed. Another important use for contact lenses is in the patient with unilateral aphakia. As you know, when a lens has been removed from one eye while the other eye retains good vision, binocular vision is impossible because of the disparity in the size of the images formed by the two eyes. The corneal lens allows binocular vision in many of these patients. Many patients who have had cataracts removed from both eyes prefer the contact lens because it enables them to dispense with a pair of heavy glasses and allows them a much wider field of vision.

In addition to their function in restoring vision contact lenses are desired by many people so they will not have to wear spectacles. The use of contact lenses in correction of high refractive errors is a more personal question and is not routinely recommended by the physician since their use is primarily for cosmetic reasons or for functional reasons by athletes, actors, models and others who prefer to dispense with glasses for certain purposes but must have adequate vision. Of this group, our experience agreed substantially with previous reports that not more than 25 to 30 per cent could wear lenses satisfactorialy. Any contact lens is an ocular foreign body and many factors enter into the ability to develop a tolerance to this foreign body. Even strong motivation will not overcome extreme hypersensitivities, allergies, etc. However refinements in manufacturing and fitting technics in the past year have increased the percentage of successful use to at least 75 per cent. What appeals to me as sensible management of these patients is the statement which a neighboring physician makes to his patients. "If you want to gamble $200.00 on the possibility of your being able to wear these lenses, your chances of doing so are about one in three." The fitting can then proceed with no false illusions.

This is the largest group of patients who want contact lenses and it is in this field that there has been much commercial exploitation with misleading advertising. While it is true that newer technical developments are increasing the number of successes in this group these lenses will never replace conventional glasses for many reasons. Of the many factors which prevent satisfactory use of contact lenses one of the most frequent is my experience has been the presence of lenticular astigmatism, which was present in amounts of one diopter over in nearly 10 per cent of a series of 237 patients. This is an astigmatic refractive error which is not manifest until the contact lens is in place and is not correctable by the lens. Other contra-indications are, small refractive errors, early presbyopes, patients with ocular allergies or infections, degenerated (phthisical) eyes and those with hypersensitive or fragile tissues.

For patients with corneal pathology who cannot be benefitted with corneal lenses, keratoplasty or corneal transplant is sometimes helpful. This operation involves replacing diseased or opaque corneal tissue with clear corneal tissue from a donor eye. Recent developments in technic have placed this operation in the realm of any competent eye surgeon, and it is rapidly becoming more widely used. The principal difficulty with this type of surgery is obtaining an adequate

supply of donor material. Keratoplasties are of two general types, penetrating, involving the full thickness of the cornea, or lamellar, involving only the superficial layers. Penetrating grafts are always partial since it has been impossible to graft the entire cornea and maintain clarity of the graft. Lamellar grafts may be partial or involve the entire cornea but the visual results are not as good as with penetrating grafts. They are principally used as therapeutic procedures in intractable corneal disease or in extensive corneal pathology where penetrating grafts are contra-indicated.

Corneal transplantation has been attempted for more than 150 years. Successful cases were reported 65 years ago but in only rare instances. The usual procedure is to remove a disc of opaque corneal tissue with a trephine and suture a similar disc of clear cornea from a donor eye into its place with direct sutures. Unfortunately, surgery is not indicated in many cases of corneal opacities and the prognosis varies greatly with each eye depending on the pathology present. The most favorable cases are those with keratoconus or central scars surrounded by clear cornea, such as a result from trauma, disciform keratitis, or familial corneal degenerations. Some successful results are obtained in corneal degenerations. Some successful results are obtained in corneal dystrophies, but improvement is seldom noted with extensive calcareous scars particularly if much vascularization is present. The use of Beta radiation both pre- and post-operatively in controlling vaccularization has improved the prognosis in many patients. Keratoplasty is definitely contra-indicated in the presence of glaucoma or active ocular inflammation of any kind.

As mentioned, the principal problem encountered in corneal surgery is an adequate supply of donor material. In the larger centers this problem has been met by the establishment of so-called Eye Banks. Actually they are not banks in the sense of storing material, since eyes should be used within 48 hours after removal, so the Eye Banks function chiefly as distribution centers. Most eyes are obtained from donors who have willed their eyes for this purpose before death. They must be removed under conditions of surgical asepsis within five hours after death, and kept in sterile containers in the refrigerator until used. In this state, when patients who need corneas are not available, the eyes are sent immediately to the Eye Banks in Chicago or New York so that none are wasted. After use these eyes are sectioned and used in research studies. Many valuable contributions to the literature have resulted from these studies. Persons wishing to donate their eyes after death often request information from their family physician as to the proper procedure. Forms can be obtained on request through the Oklahoma State Medical Association Offices, or the Secretary of the Oklahoma City Academy of Ophthalmology and Otolaryngology. It is estimated that a least ten thousand Americans could probably be made to see again through the corneal graft operation, but the only way these people will be given the opportunity to see is through the generosity of the many donors who bequeath their eyes for this purpose.

To summarize—many kinds of corneal pathology which formerly doomed the patient to permanent blindness are amenable to treatment by modern techniques with contact lenses or keratoplasty. In addition to their function in restoring vision, contact lenses may be worn by many people for cosmetic reasons or for specific functions.

Corneal transplant surgery will become available to many patients only when donor eyes become available and at present the need is far greater than the supply.

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Special Article

An Analysis of Legal Problems Resulting From Practice of ARTIFICAL INSEMINATION *in Oklahoma*

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Many articles on the problems created by artificial insemination have been written by persons in the fields of religion, social sciences, medicine and law. Opinions are as varied as the authors who have considered the subject, therefore, this discussion will purposely avoid touching on the right or wrong of the practice in a moral or social sense. The purpose of this discussion is to enlighten the physicians of the State of Oklahoma as to the legal consequences flowing from this practice.

There are two commonly used terms in reference to the practice of artificial insemination. First is artificial insemination homologous, abbreviated as AIH, which is used in the case of fertile couples where there is penile or vaginal malformation. There appears to be no serious legal consequences from this practice as the husband is the lineal father of any off-spring ensuing. The physician assisting the couple would merely be bound by the same degree of professional skill and care which is required in all medical procedure.

The practice which engenders serious legal consequences is that of heterologous insemination, commonly abbreviated as AID (artificial insemination donor), and this will be the subject of our discussion.

The law relating to AID is more astonishing by its scarcity than for any other reason. There have been very few cases dealing directly with the subject and no statutory law whatever in so far as it relates to humans. It might be interesting to note, however, that the Oklahoma Legislature has passed statutes designating Oklahoma A & M College the official Oklahoma agency to "sponsor, establish, develop and execute a program of artificial insemination for the benefit of the *Livestock Industry* of the State." It would appear that however noble this project may be, *homo sapiens* should be entitled to equal time and consideration as the bovine genus.

It must here be noted that what little law there is on this subject has no controlling effect upon the Supreme Court of this State. A New York court held, on the limited point of rights of visitation by a husband in a divorce case, that he had such rights and that the child was legitimate. The Court expressly refused to pass upon the propriety of AID or the property consequences thereof. A Canadian Court in a 1921 divorce case, in a gratuitous portion of the opinion, declared AID to be adulterous. An inferior court in Illinois stated that AID, with or without consent of the husband, is contrary to public policy and good morals and constituted adultery on the part of the mother and that a child so conceived is illegitimate. Further, the court added that the husband had no right or interest in the child.

Thus, it may be seen that the law is scarce and the holdings conflicting. The difficulty of course arises from the fact AID was unknown to the common law and that the old established doctrines can not possibly be applied to this new problem without many conflicts. Therefore in this analysis of the problem, we shall merely take the Oklahoma law

as it now is and look at the legal consequences that may arise therefrom.

The two problems most likely to cause serious litigation in this field are those of legitimacy of the child and the rights of inheritance by the child from the husband of its mother. These questions are closely related yet resolving one does not answer the other.

Our courts are loath to declare a child illegitimate and the law imposes a very strong presumption that a child born in wedlock is legitimate. Yet the fact remains that children are born in wedlock who are not the issue of the husband and are, therefore, legally illegitimate. By the same token a child born of AID may well be deemed illegitimate by the courts. This is especially true if the express consent of the husband is not given for this act.

Assuming. however, that a child born of AID is legitimate in the eyes of the law and that full consent of both parents is given, we then encounter the most serious legal problem flowing from this practice. It is that of inheritance by the child from the husband.

The uninformed "parents" feeling that their new offspring stands in the place legally of a natural born child probably take no legal action to adopt the child nor provide for it by Will. They assume that it will "inherit" their property by intestate succession. This is not so.

Our descent and distribution staute is complex and long. It provides in substance that when a person dies without a Will, the estate is distributed proportionately among the surviving spouse and issue. If there be no spouse nor issue, then the estate goes to the decedent's lineal descendants.

The critical word used in the statute is issue; which has been defined by the Oklahoma Supreme Court as it relates to this statute as follows: "issue means descendants, *lineal* descendents, offspring and includes grand-children."

Thus, it may be seen that a child born of AID definitely does not meet the statutory requirements for inheritance. The obvious impact of this being that disregarding the question of "legitimacy" as such, one of these children would in all likelihood not inherit property from a husband who is not the lineal father of the child. That is, if he dies intestate, collateral heirs related in some degree of consanguinity would be able to defeat the claims of the child by AID.

From this analysis it may be seen that even should we label a child born of AID "legitimate" so as to remove the social stigmas attached to bastards, this in and of itself has no bearing whatever upon this child's rights of inheritance. The fact remains that he probably cannot inherit from the husband of his mother.

Therefore, from a legal viewpoint, any persons desiring to have a child by AID, should, in order to protect the child's right of inheritance, provide immediately for the child by Will. Further, as Wills are revocable, may be lost and are subject to attack, the husband should also legally and formally adopt the child. If these precautions are taken, the child will be protected in Oklahoma. If they are not, the consequences may be very serious for the child.

Another question which has caused much consternation among the medical and legal professions in light of some of the decisions, is that of adultery. A forceful argument can be made that AID, without express consent of the husband, constitutes adultery. If this be true, then under our statutes, the physician performing this service could be guilty of the crime as a principal, defined in our statute as follows:

> "All persons concerned in the commission of a felony, whether they directly commit the act constituting the offense or aid in abetting its commission, although not present, must be indicted, tried and punished as principals."

The theory being that if a child is born during wedlock and the husband can by clear and convincing evidence overcome the presumption of legitimacy imposed by law, then the child being illegitimate, must have been conceived by an adulterous act.

The syllogism as stated, is perfect, but in this writer's opinion, the conclusion of adultery cannot fit the statutory definition in Oklahoma.

Our statute reads: "adultery is the unlawful, voluntary sexual intercourse of a married person with one of the opposite sex."

Thus, it may be seen, that to perpetrate the crime of adultery in Oklahoma, there must be an act of voluntary sexual inter-

course. As AID is unknown to the common law and also to statutory law, we must therefore indulge in a bit of sematology. Black's Law Dictionary, 3rd Edition, defines sexual intercourse as "carnal copulation of male and female implying actual penetration of the organs of the later." We are thus lead to the definition of carnal, which Black defines as "of the body, relating to the body; fleshy; sexual." Webster then defines copulation as: ". . . the embrace of the sexes in the act of generation."

Thus, it may seen that sexual intercourse basically implies the bodily or fleshy *embrace* of the sexes in the act of generation.

It would appear that an elaborate analysis of the cases and policy concerning adultery is unnecessary viz, AID is not adultery in Oklahoma.

What our courts will do with this problem, if and when it is presented to them, would of necessity be conjectural; however, reason would indicate that the criminal aspect of adultery resulting from AID can be discounted as to the physician, donor and wife.

The problems outlined above represent the most obvious legal consequences flowing from the practice of AID. The liability of a physician performing this function remains, however, an unknown factor. It is possible that he could also be charged with assault and battery or rape and be susceptible to civil suits in the nature of malpractice cases for injury in childbirth, etc. However, the physician can in all probability protect himself by obtaining written consent of both husband and wife and also of the donor. He should be sure that there are kept as confidential records, accessible only to him. Further, the d o n o r should remain anonymous and so agree in writing.

By being cautious and maintaining that degree of medical skill and care required to prevent malpractice suits, a physician probably has less to fear from personal legal liability than he does from the possible ill feeling of a family who although perfectly happy with their child and the medical services rendered, become entangled in the morass of archaic law as previously set forth. Therefore, a physician certainly should acquaint himself with these legal problems and his patients should also be made aware of them.

By way of conclusion, this writer would like to point out that the only sure solution to the problem is for the public, medical and legal professions to decide if AID is desirable and if so . . . to insist on legislation which will remove all doubt in this most controversial and confusing subject.

New IRS Regulations Gives Tax Advantage for Physician

The U.S. Internal Revenue Service recently issued a regulation which is important to physicians.

Efforts over a long period of time by the A.M.A. Law Department to get the Internal Revenue Service to issue a regulation permitting physicians to deduct their expenditures in taking postgraduate "refresher" courses have finally paid off.

The regulation, which went into effect on August 9, provides that expenditures for education are deductible if they are for a "refresher" or similar type of course taken to maintain the skills directly and immediately required by the physician in his employment or business. An educational course to be covered should be designed for established medical practitioners to help them keep abreast of current developments in the profession; it should be of short duration; it should not be taken on a continuing basis and should not carry academic credit. Education designed to prepare the practitioner to enter a specialty will not be acceptable.

When a physician travels away from home primarily to obtain "refresher" education, his expenditures for travel, meals and lodging while away from home are deductible. However, expenses for personal activities such as sightseeing, social visiting or entertaining, or other recreation will *NOT* be allowed.

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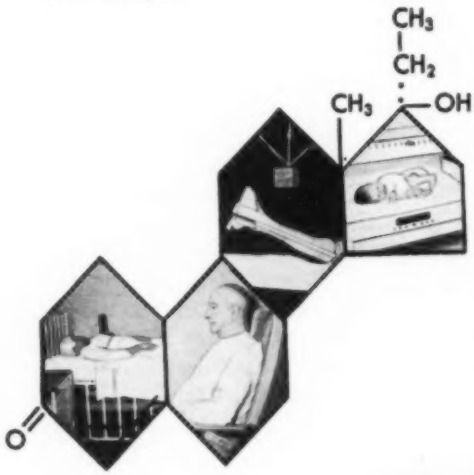

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Association Activities

PRESIDENT'S LETTER

Well the "hootin and hollerin" of our own political campaign is about over and when one scans the headlines from over the world, it appears that there is the same basic falseness in every political system, today. For instance; take a belief, a deep and delightful belief like the importance of the human being, and the importance of giving him enough freedom and helping him find enough responsibility to keep growing as a human being (the belief all religions are based on) and then separate that tenet from life and values, from integrity and honor, and tie it up with common things and with the way people are making their money at the moment; and then let a gang of avaricious men, armed with an atom, begin to manipulate all this together to get power for themselves—well, you've got a hell of a mess. That is what is happening across the face of the world today. Every political system uses men and their real needs and warm beliefs to build party power.

Don't get me wrong, I believe in government, but it should be a government of human beings by human beings for human needs and aspirations. The word DEMOCRACY is still descriptive enough for me. But the people have got to put the politician in his place. He needs to be told he has no divine rights (there is nothing divine about his political party either). Let's face it, party loyalty is a verbal switch-blade used by the little and big bosses to force us to accept a questionable situation our intelligence and conscience should reject. The politicians are of the opinion that if they are caught breaking a moral or legal law, the only punishment they need fear is a moving of their office, either up or down stairs or across the street, and—business as usual. Public servants—they call themselves (thank God there are a few). They run like the wind for office, either for monetary gains, political prestige or to inflate their ego. If they are caught (they can be) defrauding the people of this country, let them have a fair and impartial trial, but (the legal profession take note) let the trial also be fair and impartial for John Q. Public, he pays the tariff. I have a deep respect for the true statesman, but you could dump all the politicians on a boat and sink it and nothing would happen except folks might have a long deserved night's sleep.

But how can you get the voters of this country to believe they are important and that they must assist in the running of this country. It's a king size job, alright, and the most important one of our day. We must seriously consider the qualities of the candidates. Do they have honesty and integrity? Are they basically and fundamentally sound, solid Americans? Forget other so-called qualifications and party ties. The M.D.'s of this state who are qualified for political offices are derelict in their duties if these attributes are allowed to wither on the vine. Messes are never cleaned up by the tenants, the real owners of this country must do the job. "Busy," you say; if we don't do it in our individual way, then some smart politician is going to convince us it is easier, "just to leave it to him" to settle the world's problems.

Tom McClure M.D.

President

"Home Remedy" or Professional Service?

Few families today rely on home remedies when illness strikes. And that's as it should be . . . the treatment of the sick and injured should be left to the men and women who have dedicated their lives to the medical profession and the scientific treatment of the sick and injured.

However, when it comes to another important field of service to mankind—estate planning—it is unfortunate that many doctors still rely on the "home remedy." Instead of consulting those who are dedicated to estate planning, they leave the handling of their estates to well-meaning but untrained and inexperienced relatives or friends, or simply use the "home remedy" themselves.

According to an article in the American Medical Association letter of October 18, 1955:

"Only one doctor in eight survives his wife."

"One out of three physicians left no will."

"Expenses of settlement of the estates studied ranged from a minimum of 13% to as much as one-third of the total estate."

Don't delay another day, Doctor! For the sake of your family's welfare, let our Trust Department discuss with you immediately the urgent matter of the transfer of your estate in the event of your death. Call REgent 6-1531 now and ask for our Mr. Scott. He'll be happy to call on you at your convenience.

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Tulsa County Medical Society To Hold Founders Day Clinic

Oklahoma physicians are invited to attend the Fourth Annual Founders Day Clinic of the Tulsa County Medical Society in Tulsa, October 31-November 1, 1956. Four nationally known medical personalities have been scheduled as visiting distinguished guest speakers.

Robert M. Shepard, Jr., M.D., Chairman, has announced the following will appear.

Otto Saphir, M.D., Chairman of the Department of Pathology, Michael Reese Hospital, and Professor of Pathology, University of Illinois School of Medicine, Chicago, Illinois.

Walton R. Akenhead, M.D. Professor and Chairman of the Department of Medicine, Louisiana State University School of Medicine, New Orleans, Louisiana.

A. Buford Word, M.D., Associate Professor of Obstetrics and Gynecology, University of Alabama School of Medicine, Birmingham, Alabama.

Lawrence H. Strug, M.D., Professor of Surgery, Louisiana State University School of Medicine, New Orleans, Louisiana.

The annual event honoring the founders of the Society is jointly sponsored by the Tulsa Academy of General Practice, the Oklahoma Division of the American Cancer Society, and the Tulsa County Heart Association. It has been approved for formal postgraduate credit in Category One by the American Academy of General Practice.

The Clinic will open Wednesday evening, October 31, with a dinner at the Tulsa Club. Doctor Word will discuss "Congenital Absence of the Vagina." This will be the W. Albert Cook lecture in honor of Doctor W. Albert Cook, first President of the Society in 1907. Doctor Cook will be a special guest.

On Thursday November 1, a day long session will be held at the Blue Cross-Blue Shield Auditorium at 1215 South Boulder.

The program is as follows:

10:00 a.m.—Registration and Coffee
10:30 a.m.—*Cardiac Emergencies*, Walton R. Akenhead, M.D.
11:15 a.m.—*Surgical Management of Benign Lesions of the Colon and Rectum*, Lawrence H. Strug, M.D.
12:00 Noon—Luncheon
1:00 p.m.—Clinical Pathological Conference, Otto Saphir, M.D., and Walton R. Akenhead, M.D.
2:15 p.m.—*Abnormal Utering Bleeding*, A. Buford Word, M.D.
3:00 p.m.—Coffee and Refreshments
3:15 p.m.—*Esophageal Hiatus Hernia*, Lawrence H. Strug, M.D.
4:00 p.m.—*Spread of Human Cancer*," Otto Saphir, M.D.
4:45 p.m.—Adjournment

There is no charge for the Clinic, and all medical doctors are urged to attend. Dinner will be $5.00, including social hour, and luncheon will be $1.50. Tickets may be ordered from the Tulsa County Medical Society, B-9 Medical Arts Building, Tulsa, Oklahoma. Hotel reservations are available upon request of the Society.

Grand Rounds Telecast Offered Oklahoma Physicians

An invitation has been extended to Oklahoma physicians by the Upjohn Company to attend a closed circuit, live telecast entitled "The Borderlines of Cancer; Problems in the Management of Dubious Lesions," which is 90 minutes in length.

The telecast will be presented Wednesday, October 17, in the Venetian Room of the Skirvin Hotel, Oklahoma City, at 7:00 p.m. It will emanate from Tufts University School of Medicine and the New England Medical Center in Boston.

One of the outstanding features of this program is the good geographic representation of the panel. The panelists and their affiliations are:

Lauren V. Ackerman, M.D., Washington University School of Medicine, St. Louis, Mo.

Edwin B. Astwood, M.D., Tufts University School of Medicine, and the New England Center Hospital, Boston, Mass.

Henry L. Bockus, M.D., University of Pennsylvania School of Medicine, Philadelphia, Pa.

George Crile, Jr., M.D., Cleveland Clinic, Cleveland, Ohio.

Leo H. Garland, M.D., Stanford University School of Medicine, San Francisco, Calif.

Phillip J. Hodes, M.D., Professor of Radiology, School of Medicine, University of Pennsylvania.

Alton Ochsner, M.D., Tulane University School of Medicine, New Orleans, La.

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1. Cronheim, G., and Tockus, I. M.: Comparison of Sedative Properties of Single Alkaloids of Rauwolfia and Their Mixtures, Meet. Am. Soc. Pharmacol. & Exper. Therap., Iowa City, Iowa, Sept. 5, 1955.

2. Moyer, J. H.; Dennis, E., and Ford, R.: Drug Therapy (Rauwolfia) of Hypertension. II. A Comparative Study of Different Extracts of Rauwolfia When Each Is Used Alone (Orally) for Therapy of Ambulatory Patients with Hypertension, A.M.A. Arch. Int. Med. 96:530 (Oct.) 1955.

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LOS ANGELES

New Federal Loans To Health Facilities

The newest federal loan program in the health field now appears to be underway: Small Business Administration loans to proprietory hospitals, nursing homes and medical and dental clinics. Unlike the Hill-Burton Act and most other federal programs, this is not directed to non-profit organizations.

The following is a list of definitions for eligible facilities:

Hospitals are those facilities which are licensed as hospitals providing in-patient medical or surgical care of the sick or injured, including obstetrics, and which are privately owned and operated for profit.

Nursing homes are those proprietary facilities for the accommodation of convalescents or other persons who are not acutely ill and not in need of hospital care but who may require nursing care and related medical services.

Medical and dental laboratories are those proprietary units that provide services to physicians, dentists, hospitals and similar health facilities.

Under SBA regulations, any establishment seeking a loan must qualify as a small business operation. In the case of this new field, hospitals and nursing homes will be considered small business when capacity does not exceed 50 beds at time of application. All laboratories shall be considered small unless operated in connection with a proprietary hospital of more than 50 beds.

SBA makes two types of loans: direct and participating, the latter jointly with a bank. Other criteria include: (1) the applicant must show that the needed financing is not otherwise available on reasonable terms from another credit source, (2) applicant must show that the loan can be repaid out of earnings, (3) owners and operators of a facility must be experienced in their field and with enough professional training to operate in accordance with accepted standards, (4) when licensing is required by state, county or local agency, the facility must have a license in good standing or show that a license will be issued, and (5) applicant for direct loan must enclose letter from the private lender stating it is unable to make the loan.

Applications and information are available at SBA regional offices in Boston, New York, Philadelphia, Richmond, Atlanta, Cleveland, Chicago, Minneapolis, Kansas City, Dallas, Denver, San Francisco, Los Angeles, Seattle and Detroit.

Association Attends Department Of Defense Meeting in Denver

At a meeting in Denver, Colorado on August 25-26, were representatives of the majority of the states west of the Mississippi River, to hear discussions on the plans and operations of Public Law 569, passed by the 84th Congress to provide Medical Care for the Dependents of Military Personnel. This meeting followed a previous meeting in Chicago called by the American Medical Association of all of the states and territories.

Attending the Denver meeting, representing the Oklahoma State Medical Association were: H. H. Macumber, M.D., Chickasha, Chairman of the Association's Committee on Medical Service, Hospitals and Prepaid Insurance, and Dick Graham and Don Blair from the Executive Offices.

The discussions were principally devoted to the manner in which the State Medical Associations would designate their individual contracting agencies, and fiscal representatives. Based on preliminary reports by representatives of the states in attendance the Contracting Agent would be the individual State Associations with Blue Shield acting as the Fiscal Agency only.

Public Law 569 goes into effect on December 7, 1956.

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Five Physicians Receive Life Memberships

At a recent meeting of the Tulsa County Medical Society, five veteran Tulsa physicians were presented certificates of Life Membership in the Oklahoma State Medical Association.

Pictured left making the presentation is Wilkie D. Hoover, M.D., Tulsa surgeon and Councilor to the Oklahoma State Medical Association.

Left to right receiving the certificates are: Harry Dale Murdock, M.D., Delbert O. Smith, M.D., James H. Neal, Sr., M.D., Fletcher L. Nelson, M.D., and Ernest W. Reyolds, Sr., M.D. The five have a total of 243 years of practice.

East Central Society, VA Jointly Sponsor Seminar

Oklahoma physicians are cordially invited to attend the Seminar on Management of Traumatic Injuries sponsored jointly by the East Central Oklahoma Medical Society and the Veterans Administration Hospital staff, November 1, 1956 in Muskogee.

Program

Afternoon—Auditorium Veterans Administration Hospital

Welcome—D. H. Miller, M.D., Manager, VAH, Muskogee

Moderator—Chester K. Mengel, M.D., VAH, Muskogee

2:00-3:00 p.m.—*Management of Traumatic Injured*, Earl D. McBride, M.D., from the point of view of an Orthopedist (Oklahoma City); Clifford Allen, M.D., from the point of view of a Radiologist (Oklahoma City). *Comments*—Donald R. Adams, M.D., VAH, Muskogee.

3:30-4:00 p.m.—Coffee Break

4:00 p.m.—William N. Weaver, M.D., Moderator—*What's Your Diagnosis—Differential Diagnosis of Abdominal Conditions*, Hugh Bennett, M.D., Internist, VAH, Houston; George Brown, Jr., M.D., Surgeon, McAlester; Simon Dolin, M.D., radiologist, Oklahoma City.

Evening—Hotel Severs

7:30 p.m.—Dinner

8:30 p.m.—*Abnormal Bleeding Problems in Pregnancy*, Roy G. Holly, M.D., Omaha, Neb.

Five hours accreditation will be given in Catagory II for attending this program.

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OKLAHOMA CITY CLINICAL SOCIETY

October 22-23-24-25, 1956 — Biltmore Hotel

PROGRAM

Monday, October 22

9:00 a.m.—*Chronic Pelvic Pain*, Dr. Willis E. Brown, Little Rock, Ark.

9:30 a.m.—*Anesthesia Errors in Obstetrics*, Dr. Henry B. Turner, Memphis, Tenn.

10:00-10:30 a.m.—*Visit the Exhibits*

10:30 a.m.—*Interpretations of the Knee Injuries*, Dr. Edward T. Evans, Minneapolis, Minn.

11:00 a.m.—*Diverticulitis of the Colon*, Dr. Albert Behrend, Philadelphia Pa.

11:30 a.m.—*Vertigo, Dizziness and Giddiness: Their Differential Diagnosis*, Dr. Kenneth B. Corbin, Rochester, Minn.

9:30 a.m.—Specialty Lecture—*Correlation of BMR, Serum Cholesterol, I^{131} Uptake and Serum PBI in the Differential Diagnosis of Thyroid Disease*, Dr. William H. Beierwaltes, Ann Arbor, Mich.

9:30 a.m.—Specialty Lecture—*Primary Closure of the Exstrophied Bladder*, Dr. A. Keller Doss, Fort Worth, Tex.

12:15-1:40 p.m.—*Luncheon—Round Table Discussion.*

2:00 p.m.—*Ocular Emergencies*, Dr. Rufus C. Goodwin, San Francisco, Calif.

2:30 p.m.—*Radiographic Evaluation of the Genito-Urinary System*, Dr. A. Keller Doss, Fort Worth, Tex.

3:00-3:30 p.m.—*Visit the Exhibits*

3:30 p.m.—*Treatment of Hyperthyroidism*, Dr. William H. Beierwaltes, Ann Arbor, Mich.

4:30 p.m.—*The Unknown Killer of Obstetrics*, Dr. Henry B. Turner, Memphis, Tenn.

4:30 p.m.—*Thyroid Nodules, Their Significance, Diagnosis and Treatment*, Dr. Patrick J. Fitzgerald, New York City, N. Y.

2:30 p.m.—Specialty Lecture—*Common and Uncommon Causes of Hemorrhage from the Gastro-Intestinal Tract*, Dr. Albert Behrend, Philadelphia, Pa.

2:30 p.m.—Specialty Lecture—*Pertinent Observations with Reference to Bone Tuberculosis, Its Morbidity, Mortality and Modern Concepts of Its Treament*, Dr. Edward T. Evans, Minneapolis, Minn.

7:00 p.m.—*Banquet—Sponsored by the Oklahoma County Medical Society.* Guest Speaker—Harold W. Elley, Ph.D., Wilmington, Del.

Tuesday, October 23

9:00 a.m.——*Observations of the Growth Rate of Human Tumors*, Dr. Vincent P. Collins, Houston, Tex.

9:30 a.m.—*The Diagnosis and Treatment of Brachial Pain Problems*, Dr. Kendall B. Corbin, Rochester, Minn.

10:00-10:30 a.m.—*Visit the Exhibits*

10:30 a.m.—*Clinical Pathological Conference*, Dr. Patrick J. Fitzgerald, New York City, N. Y.; Dr. Gordon Meiklejohn, Denver, Colo.

9:30 a.m.——Specialty Lecture—*The Urinary Tract and the Gynecologist*, Dr. Willis E. Brown, Little Rock, Ark.

9:30 a.m.—Specialty Lecture—*The Surgical Management of Gastric Cancer Based on the Pathological Study of the Lymphatic Spread*, Dr. Gordon McNeer, New York City, N. Y.

12:15-1:40 p.m.—*Luncheon—Round Table Discussion*

2:00 p.m.—*Shoulder Cuff and Shoulder Injuries*, Dr. Edward T. Evans, Minneapolis, Minn.

2:30 p.m.—*Surgical Jaundice*, Dr. Albert Behrend, Philadelphia, Pa.

3:00-3:30 p.m.—*Visit the Exhibits*

3:30 p.m.—*Treatment of Hypothyroidism*, Dr. William H. Beierwaltes, Ann Arbor, Mich.

4:00 p.m.—*Prophylactic Gynecology*, Dr. Willis E. Brown, Little Rock, Ark.

4:30 p.m.—*Urological Problems of Interest to the Man in General Practice*, Dr. A. Keller Doss, Fort Worth, Tex.

2:30 p.m.—Specialty Lecture—*The Influence of Soaps and Detergents on Skin Physiology*, Dr. Donald J. Birmingham, Cincinnati, Ohio

2:30 p.m.—Specialty Lecture—*Tidy Obstetrics*, Dr. Henry B. Turner, Memphis, Tenn.

7:00 pm.—*Annual Clinic Dinner Dance*, Persian, Room, Skirvin Tower Hotel; Dr. Harry Wilkins, President, Presiding

Wednesday, October 24

9:00 a.m.—*Contact Dermatoses Produced by Over-Treatment*, Dr. Donald J. Birmingham, Cincinnati, Ohio

9:30 a.m.—*Problems in Chomotherapy*, Dr. Gordon Meiklejohn, Denver, Colo.

10:00-10:30 a.m.—*Visit the Exhibits*

10:30 a.m.—*Retarded Growth and Development in Infants and Children*, Dr. John F. Crigler, Jr., Boston, Mass.

11:00 a.m.—*Management of Common Nasal Complaints*, Dr. Joseph H. Ogura, St. Louis, Mo.

11:30 a.m.—*Melanoma—Treatment and End Results*, Dr. Gordon McNeer, New York City, N. Y.

9:30 a.m.—Specialty Lecture—*"Corneoscleral Sutures*, Dr. Rufus C. Goodwin, San Francisco, Calif.

9:30 a.m.—Specialty Lecture—*X-Ray Diagnosis of the Aging GI Tract*, Dr. Vincent P. Collins, Houston, Tex.

12:15-1:40 p.m.—*Luncheon—Round Table Discussion*

2:00 p.m.—*Use of Steroid Hormones in Various Allergic Disorders*, Dr. Ethan Allan Brown, Boston, Mass.

2:30 p.m.—*Accelerated Growth and Development in Infants and Children*, Dr. John F. Crigler, Jr., Boston, Mass.

3:00-3:30 p.m.—*Visit the Exhibits*

3:30 p.m.—*The Treatment of External Ocular Diseases*, Dr. Rufus C. Goodwin, San Francisco, Calif.

4:00 p.m.—*Advances in Viral Diseases*, Dr. Gordon Meilkejohn, Denver, Colo.

4:30 p.m.—*Hoarseness: Diagnosis and Treatment*, Dr. Joseph H. Ogura, St. Louis, Mo.

2:30 p.m.—Specialty Lecture—*Basic Problems of Drug Allergy*, Dr. Ethan Allan Brown, Boston, Mass.

2:30 p.m.—Specialty Lecture—*A Review of Recent Concepts Regarding the Physiology of the Brain Stem Reticular Formation*, Dr. Kendall B. Corbin, Rochester, Minn.

6:30 p.m.—*Specialty Group Dinners*

Thursday, October 25

9:00 a.m.—*The Pulmonary Cripple*, Dr. Ethan Allan Brown, Boston, Mass.

9:30 a.m.—*X-Ray Examination of the Chest in Pediatrics*, Dr. Vincent P. Collins, Houston, Tex.

10:00-10:30 a.m.—*Visit the Exhibits*

10:30 a.m.—*Hobbies, Hazards and Skin Health*, Dr Donald J. Birmingham, Cincinnati, Ohio

11:00 a.m.—*End Results in the Treatment of Gastric Cancer*, Dr. Gordon McNeer, New York City, N. Y.

9:30 a.m.—Specialty Lecture—*The Present Concept of Adrenal Steroid Biosynthesis and Its Importance in Understanding and Treating Patients with Adrenal Hyperplasia*, Dr. John F. Crigler, Jr., Boston, Mass.

9:30 a.m.—Specialty Lecture—*Present Status of Cancer of the Larynx*, Dr. Joseph H. Ogura, St. Louis, Mo.

12:15-1:40 p.m.—*Luncheon—Round Table Discussion*

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Hospital Liable For Employee 'Medical Acts'

The "Medical News" published by the Ciba Pharmaceutical Products Inc. stated in its September 10, 1956 issue that the New York State's Court of Appeals has ruled that a non-profit hospital is liable for an employee's negligence in certain "medical acts" as well as "administrative acts."

Up to now New York courts have held that a nonprofit hospital may not be held liable for negligence in medical acts performed under supervision of doctors and nurses.

In the decision recently announced reversing a ruling of the Appellate Division of the State Supreme Court in the much discussed case of Berg vs. New York Society for the Relief of the Ruptured and Crippled, the Court of Appeals, as the State's highest court, has further reduced the area of immunity from malpractice actions for nonprofit hospitals.

Award Upheld

The New York Court of Appeals upheld the original trial court's finding that the defendant hospital was liable for a technician's error in a blood test prior to a transfusion. It affirmed the lower court's award of a judgment for $17,500 to the plaintiff, Mrs. Rose S. Berg, for injuries resulting from transfusions of incompatible blood based on the erroneous blood analysis.

The technician had mistakenly reported her blood type as A-Rh positive, when it was in fact A-Rh negative. Complications during and after the ensuing pregnancy included stillbirth and a vaginal hysterectomy.

In its opinion, which was unanimous, the Court of Appeals ruled that the blood test was a "medical act in the sense that it was preparatory to a transfusion."

Narrows Gap

This opinion thus has the effect of narrowing, without fully closing, the gap between legal liability and immunity under the legal definitions of "medical acts" and "administrative acts." Both are legal terms, and even veteran jurists do not agree on the definitions.

The trial court, for example, held the blood test to be an *administrative* act, for which the hospital was liable. The Appellate Division reversed this finding on grounds that the test was a *medical* act, for which the hospital was not liable.

The Appellate Division said in part: "As we read the prevailing authorities in this state, the negligent act in this case bears a sufficiently direct and immediate relation to the care and treatment specified for the plaintiff by her own physician to require that the judgment below be reversed and the complaint dismissed."

Court's Opinion

In reversing this decision, the Court of Appeals ruled that although the test was a "medical act," it was performed "not by a physician or nurse but by a technician who was employed and paid by the hospital and who was so far short of professional status or attainments that only four to six weeks training was necessary for her job. She was no independent practitioner of a learned profession . . . but a salaried employee."

The opinion, written by Justice Charles S. Desmond, said in part:

"Not only do they (modern hospitals) furnish room and board to patients but they sell them services which are 'medical' in nature and, though furnished on physicians' orders, are performed wholly by and under the control of the hospitals' salaried staffs.

"What reason compels us to say that of all employees working in their employers' businesses (including charitable, educational, religious and governmental enterprises) the only ones for whom the employers can escape liability are the employees of hospitals?"

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Committee on Medical Service Work on Public Law 569

The Committee on Medical Service, Hospitals and Prepaid Insurance, composed of: H. H. Macumber, M.D., Chickasha, Chairman, Horton E. Hughes, M.D., Shawnee, J. E. Highland, M.D., Miami, Henry T. Russell, M.D., Enid, C. M. Bielstein, M.D., Oklahoma City, Gerald Rogers, M.D., Oklahoma City and Walter E. Brown, M.D., Tulsa, has been designated by President H. M. McClure to do the necessary research and planning to implement Public Law 569, commonly referred to as Medical Care for the Dependents of Military Personnel.

The Committee has already had two meetings, and has made a preliminary report to the Council.

Council Approves Preliminary Report

The Committee made four recommendations to the Council at the Council Meeting on September 9, 1956. These recommendations were:

1. That the Association participate in this program.
2. That a Contract of no more than one year duration be entered into.
3. That the Oklahoma State Medical Association act as its own Contracting Agency.
4. The Blue Shield plan act as the fiscal agency.

Fee Schedule

Under the provisions of the Act, the medical care to be rendered dependents will be on a service basis, and the Association must negotiate a fee schedule with the Department of Defense. The Nomenclature for the Fee Schedule that has been prepared by the Department of Defense covers over 1400 schedules.

The Council further authorized the present studying Committee to continue its work, including the working out of a Fee Schedule which subsequently, will be submitted to the Council.

County Society Sent Questionnaire

Concerning the problem of fees, all County Medical Society Secretaries were sent a questionnaire the latter part of August, and over 75 per cent of the County Societies have already returned the questionnaires.

Cancer Conference Speakers Named

The Oklahoma Division of the American Cancer Society announces their annual meeting to be held at the University of Oklahoma Medical School auditorium on December 8, 1956.

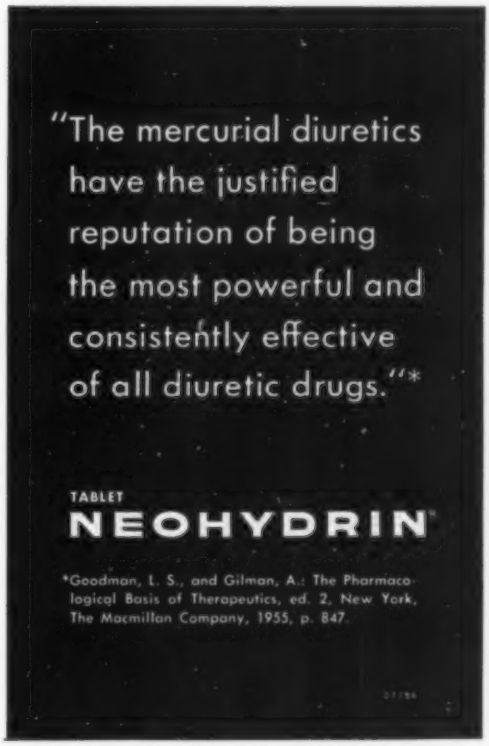
Joe Parker, M.D., chairman of the Professional Education Committee has announced that the following out-of-state physicians will be among the guest speakers on the program:

Roger A. Harvey, M.D., head of the Department of Radiology at the the University of Illinois College of Medicine.

Hayes Martin, M.D., attending Surgeon at Memorial Hospital and Assistant Professor of Clinical Surgery at Cornell University Medical College, New York City.

John R. Schenken, M.D., Professor of Pathology and Bacteriology at the University of Nebraska Medical School, Omaha, and Director of Laboratories at Nebraska Methodist Hospital.

Richard W. TeLinde, M.D., Professor of Gynecology at Johns Hopkins Medical School, Baltimore, Md.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*Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 2, New York, The Macmillan Company, 1955, p. 847.

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formula: Each tablet of METRETON provides 2.5 mg. of METICORTEN (prednisone), 2 mg. of CHLOR-TRIMETON maleate (chlorprophenpyridamine maleate), and 75 mg. ascorbic acid.

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Doctor Finis W. Ewing Subject of Special Article

The *Muskogee Daily Phoenix*, in its September 9, issue carried a special article concerning the life history of Doctor Ewing, who was the President of the Oklahoma State Medical Association in 1941-42.

The article told interesting anecdotes in Doctor Ewing's life and the honors that had come to him over the years by virtue of his public service records, and his contribution to his profession.

He is an honorary life member of the Masonic Bodies, and the Shrine, and the Oklahoma State Medical Association. He is a Charter Member of the Muskogee Kiwanis Club.

Doctor Ewing was born at Cornelia, Missouri, January 10, 1876. Dr. and Mrs. Ewing live at 2802 West Broadway, Muskogee, Oklahoma.

OSMA Woman's Auxiliary Holds Annual Fall Meeting

The Annual Fall Conference of the Woman's Auxiliary to the Oklahoma State Medical Association was held in the headquarters of the Oklahoma State Medical Association on October 5, 1956.

Presiding over the meeting was Mrs. L. Gordon Livingston of Cordell, President of the organization.

Attending the meeting were the Presidents of the County Auxiliaries, the Officers of the State Auxiliary, Chairmen of the Committees and many Members-at-large.

The meeting included reports from the Officers and the Committees, as well as panel discussions.

Over fifty members were in attendance and the meeting constituted the largest meeting so far held in the new Executive Offices.

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ATARAXOID exerts the anti-rheumatic, anti-inflammatory activity of STERANE distinctly superior to previous steroids, effective in radically reduced dosage, and with minimal disturbance of electrolyte and fluid metabolism.

The ataractic effect is a central neuro-relaxing action – the result of a marked cerebral specificity – free of mental fogging and devoid of any major complications: no liver, blood or brain damage. This peace-of-mind component is also used in the lowest dosage range.

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simultaneously controls the symptoms and the apprehension

In Rheumatoid Arthritis, other collagen diseases, bronchial asthma and inflammatory dermatoses

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Federal Funds for Hospital Construction Receive Approval

The Oklahoma State Department of Health, State Agency for the Federal-Aid Hospital Construction Program (Hill-Burton), has announced final approval at the state level of the Revised State Plan and the Project Construction Schedule for the fiscal year 1956-57, following approval by the State Hospital Advisory Council on September 16, 1956, and affirmed by the State Board of Health on September 23, 1956.

G. F. Mathews, M.D., State Commissioner of Health, said that the construction schedule was determined on a basis of relative need, utilization and eight other priority factors, including the nature and scope of the proposed projects and ability of the sponsor to construct, maintain and operate.

Sponsors for each project on the 1956-57 schedule will be allowed 90 days to indicate whether they desire to exercise their priority rights, and an additional 90 days to satisfy the financial requirements for the sponsor's share of costs if they wish to proceed with the project.

Approved projects for 1956-57 under Part C (the original Hill-Burton program) include the following:

Hillcrest Medical Center, Tulsa. 94-bed addition. Total estimated cost, $2,000,000; Federal allotment, $283,333.67.

St. Anthony's Hospital, Oklahoma City. 120-bed addition. Total estimated cost, $1,392,044.84; Federal allotment, $137,500.00.

Muskogee City-County Health Department, Muskogee. Heating system. Total estimated cost, $10,500; Federal allotment, $5,250.

Jackson County Health Deparament, Altus. New public health center. Total estimated cost, $50,000; Federal allotment, $25,000.

University Hospital, Oklahoma City. X-ray, therapy and addition to Out-Patient Department. Total estimated cost, $510,782; Federal allotment, $204,312.80.

Oklahoma Osteopathic Hospital, Tulsa. 60-bed addition. Total estimated cost, $647,859; Federal allotment, $259,143.

Grady County Health Center, Chickasha. New 75-bed general hospital and public health center. Total estimated cost, $1,376,800; Federal allotment, $510,000.

Approved projects for 1956-57 under Part G (1954 Medical Facilities Act, covering Diagnostic and Treatment Centers, Rehabilitation, Nursing Homes, and a renewed emphasis on Chronic Disease Hospitals) include:

St. Anthony's Hospital, Oklahoma City. Out-Patient Department. Total estimated cost, $147,955.16; Federal allotment, $73,977.58.

Oklahoma Osteopathic Hospital, Tulsa. Out-Patient Department. Total estimated cost, $147,955.16; Federal allotment, $73,977.58.

Comanche County Memorial Hospital, Lawton. New 40-bed chronic disease wing. Total estimated cost, $300,000; Federal allotment, $150,000.

Oklahoma Osteopathic Hospital, Tulsa. New 26-bed chronic disease wing. Total estimated cost, $176,901; Federal allotment, $88,450.

Mary Hurley Nursing Home, Coalgate. New 40-bed nursing home. Total estimated cost, $200,000; Federal allotment, $100,000.

All above proposed projects are subject to final approval of the Revised State Plan for 1956-57, and the Project Construction Schedule, by the Surgeon General, Public Health Service.

Projects approved *tentatively* for 1957-58, contingent on Federal appropriations and unknown projects of higher priority, include:

Murray County Health Center, Sulphur. New 25-bed general hospital and public health center. Total estimated cost, $425,000; Federal allotment, $173,000.

Bristow Health Center, Bristow. 16-bed addition. Total estimated cost, $92,055.90; Federal allotment, $36,822.

Tahlequah City Hospital, Tahlequah. Addition. Total estimated cost, $83,000; Federal allotment, $33,000.

Nowata General Hospital, Nowata. 17-bed addition. Total estimated cost, $150,000; Federal allotment, $60,000.

Thomas Memorial Hospital, Thomas. 10-bed addition. Total estimated cost, $57,985; Federal allotment, $23,194.

Cordell Memorial Hospital, Cordell. New 22-bed general hospital. Total estimated cost, $311,000; Federal allotment, $86,000.

Sapulpa City Hospital, Sapulpa. New 35-bed general hospital. Total estimated cost, $511,000; Federal allotment, $204,640.

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- handy 15 cc. plastic dropper-bottle

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(Excellent, too, for stimulating appetites of the elderly patient!) Dosage: 0.5 to 1 cc. (10-20 drops) daily. Each cc. (20 drops) contains:

l-Lysine	300 mg.
Vitamin B_{12}	25 mcgm.
Thiamine (B_1)	10 mg.
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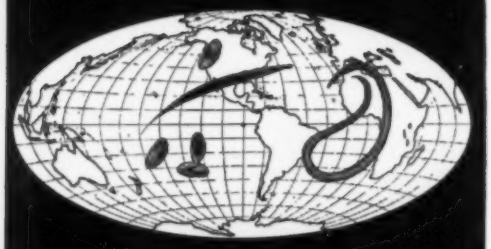

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Physicians To Attend Seattle Meeting

The 10th AMA Clinical Meeting will be held in Seattle, November 27-30. This is the second time the meeting has been held on the West Coast. The previous West Coast meeting was held three years ago in Los Angeles.

Scientific Program

Major emphasis at this Clinical Meeting will be in the fields of Obstetrics, Fractures, Heart Disease, and Psychiatry. Other scientific topics will include Arthritis, Dermatology, Gastro-Intestinal Diseases, Neurology, Gynecology, Pediatrics, Pulmonary Diseases, and Surgery.

Oklahomans Will Attend

Those who will attend from Oklahoma include, Doctors McClure, President of the Oklahoma State Medical Association, Wilkie D. Hoover, Tulsa, John F. Burton, Oklahoma City, Delegates to the AMA, and E. H. Shuller, McAlester, and Malcom E. Phelps, El Reno, Alternate Delegates to the AMA.

Oklahoma Physicians who may be going to Seattle for the Meeting can make advance registration by writing the AMA.

For hotel reservations, write direct to the Seattle Hotel Association, 315 Seneca, Seattle, Washington, and designate time of arrival and their desires concerning hotel rate brackets. Headquarters for the housing of the Delegates of the AMA will be the Olympic Hotel where no reservations are available.

SW Cancer Conference Will Meet in Ft. Worth

Mark October 26 and 27 on your calendar as a reminder of the Southwestern Cancer Conference to be held in Fort Worth, Texas.

Among the visiting distinguished speakers will be:

W. A. D. Anderson, Professor of Pathology, Miami University; W. Emory Burnett, Professor of Surgery, Temple University and Don Childs, Professor of Radiology, University of Minnesota.

This conference is sponsored jointly by the Tarrant County Medical Society and the American Cancer Society.

Oklahoma Physician To Appear On Endocrinology Program

Henry H. Turner, M.D., of Oklahoma City, past president of the Association, will participate on two panels at the 8th Postgraduate Assembly in Endocrinology and Metabolism to be held October 22-27, 1956 at the Texas Medical Center in Houston, Texas.

Twenty-three clinicians and investigators from various parts of the country will compose the faculty for the Assembly program which is jointly sponsored by the Endocrine Society, M.D. Anderson Hospital and Tumor Institute, where it will be held, and The University of Texas Postgraduate School of Medicine.

The course is designed to cover the main aspects of diagnosis and therapy in the field of endocrinology and metabolism for the physician in general practice and for those in other specialties who wish to have a general knowledge of this rapidly growing field.

Registration is limited to 100. Tuition fee is $100.00 which will be paid to The Endocrine Society for assembling the out-of-state faculty.

If further information is desired please write to: Office of the Dean, The University of Texas, Postgraduate School of Medicine, Texas Medical Center, Houston 25, Texas.

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In clinical trials, over 80% of cases have been cleared of the infection by one course of treatment with 'Antepar.'

Rumbalo, T. S., Gustina, F. J., and Oleksiak, R. E.: J. Pediat. 44:386, 1954.

White, R. H. R., and Standen, O. D.: Brit. M. J. 2:755, 1953.

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"Ninety per cent of the children passed all of their ascarides . . ."

Brown, H. W.: J. Pediat. 45:419, 1954

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6 Ounces Daily Supply The Following:

Complete Protein	19 gms.
Iron	10 mgs.
Calcium	264 mgs.
Vitamin B_1	1.2 mgs.
Vitamin B_2	.79 mgs.
Niacin	9 mgs.

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Sold Fresh At Leading Grocers'... Costs More Than Ordinary White Bread, But About Cuts The Cost Of The Above Nutrients In Half Compared With The Same In A Powdered Supplement Form

"Diet. The basal diet of the aged should be designed according to the same principles as applied to other adult groups. However, because of the frequently low total consumption of food, it is wise to encourage a liberal intake of protein and other protective foods. Three or four meals per day should be planned. The aged tolerate large meals poorly. Foods with soft bulk should be included. Fats should be restricted according to each patient's tolerance. In general, it is wise to avoid fried and fatty foods." Chapter 13—Drs. William H. Sebrell, Jr., and James M. Hundley, *Geriatric Medicine*, Edited by Edward J. Stieglitz M.S., M.D., F.A.C.P., Third Edition, J. B. Lippincott Company.

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KARO® SYRUP . . . meets the accelerated nutritional requirement of early infancy

The high caloric requirements of infants, coupled with the fact that they have little ability to digest starchy food, are indications for the use of an easily digested carbohydrate milk modifier.

Karo places a minimum demand upon the digestive system during the first weeks of life. Even premature babies thrive on Karo because this easily digested, completely utilized, fluid mixture of dextrins, maltose and dextrose does not induce flatulence, colic, fermentation or allergy.

Karo permits easy adjustment of formula to meet the accelerated nutritional demand during the early months of rapid growth. Mothers will appreciate the ease of making formulas containing Karo, plus its ready availability and economy. Light or dark Karo syrup may be used interchangeably with cow's milk or evaporated milk and water. Each ounce yields 120 calories.

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Sears-Roebuck Plan Offers Financial Assistance to Doctors

For the past two years the American Medical Association has been cooperating with the Sears-Roebuck Foundation in a program which is hoped to fill the gap in providing improved medical care where there is the greatest need.

The Foundation makes an annual grant of $125,000 which is placed in a revolving fund and made available to physicians desiring to establish practices or to improve existing facilities.

Individual physicians may receive unsecured loans in amounts up to $25,000. The primary objective of the program is to improve medical services and facilities in suburban and rural areas.

The interest rate ranges from zero to six per cent over a 10 year period depending on the rapidity of repayment. There is no charge for administration of the program as this is met by additional Foundation Funds.

To date the Foundation has made 22 loans, affecting 33 physicians, in 13 states totaling $172,000. Oklahoma has received four of these loans. So that applications may be processed efficiently, there are two cut-off-dates, October 1 and April 1. Applications received prior to October 1 will be decided upon no later than December 15. Applications received between October 1 and April 1 shall be decided upon no later than June 15.

Applications should be sent to the office of the region in which the proposed medical plan is to be established. Detailed information concerning this plan may be obtained from the office of the Oklahoma State Medical Association.

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Have You Heard?

ELVIN M. AMEN, M.D., Bartlesville physician left recently for San Antonio, Texas where he has been called back into the Air Force.

ROBERT G. ALLEN, M.D., Bartlesville, discussed poliomyelitis and the Salk vaccine at the Bartlesville Kiwanis Club recently.

WARREN AUSTERMAN, M.D., Konawa physician for eight years, entered the army medical corps in July as a captain.

ROBERT M. BIRD, M.D., Oklahoma City, spoke on "Medical Considerations in Splenectomy" at the regular monthly clinical conference held at the McAlester Clinic.

EUGENE BOND, M.D. has recently been discharged from the air force and is taking a surgical residency in Oklahoma City. Prior to entering service, he practiced in Fairfax.

REECE R. BOONE, JR., M.D., formerly of Montgomery West Virginia, is the new chief surgeon at the Northwest Community Hospital in Mooreland.

LEONARD H. BROWN, M.D. has recently opened offices at 430 Northwest 12 Street, Oklahoma City.

GLENN W. COSBY, M.D., Miami physician is the new chairman of the board of directors of Ottawa County's Red Cross chapter.

REX DAUGHERTY, M.D. has joined the Walker-Manning Clinic in Pawhuska. He has just completed his internship at Hillcrest Hospital, Tulsa.

JOHN DEVORE, M.D., Oklahoma City, spoke on the various types of leukemia at a Comanche-Cotton Medical Society meeting.

LEONARD P. ELIEL, M.D., head of the clinical cancer section of the Oklahoma Medical Research Foundation, has been named acting director.

JAMES MITCHELL FITE, M.D. has recently taken over duties in obstetrics and gynecology at the Fite Clinic in Muskogee.

J. DOUGLAS GREEN, M.D., Cushing, spoke on the Salk vaccine at a PTA meeting in Cushing recently.

WILLIAM T. GYLES, M.D., formerly from Rush Springs has opened his offices in Harrah.

ROBERT E. HERNDON, M.D. of the Chickasha Hospital and Clinic, has been notified of his certification by the American Board of Pediatrics.

R. A. HILL, M.D., a graduate of the University of Oklahoma School of Medicine has opened his practice in Antlers.

F. W. HOLLINGSWORTH, M.D. has resumed his practice in El Reno after three years surgical residency in surgery in Oklahoma City.

J. E. HOLLIS, M.D., Bristow, has been named chief of staff of the Bristow Memorial Hospital.

GEORGE H. JENNINGS, M.D., former Wetumka physician has opened offices for the practice of obstetrics and gynecology at the Medical Center Building in Oklahoma City.

ROGER G. JOHNSON, M.D., Frederick physician has recently joined JOE HORTON, M.D. and F. P. FRY, JR, M.D. in the Medical-Surgical Clinic, Frederick.

WILLIAM P. JOLLY, M.D., internal medicine specialist, has joined FRANK H. AUSTIN, M.D. in the Medical Center, Lawton.

E. M. POER, M.D., Mangum, was president of the Greer County Homecoming committee for a celebration held in July.

THURMAN SHULLER, M.D., McAlester, spoke on the Salk vaccine at a recent PTA meeting in that city.

GEORGE R. SMITH, JR., M.D., has set up practice at the Bassett Clinic, Cushing. Doctor Smith's practice is limited to Internal Medicine and Diseases of the Heart and Lung.

ORANGE W. STARR, M.D., practicing physician in Drumright for 41 years has recently retired.

PATRICK SHANKS, M.D., Enid, has been awarded a fellowship in the American Academy of Obstetrics and Gynecology.

HENRY H. TURNER, M.D., W. K. WEST, M.D. and E. S. LAIN, M.D. will attend the Golden Anniversary Meeting of the Southern Medical Association to be held November 12, 13, 14, 15, 1956 in Washington, D.C.

J. L. WHEELER, M.D., Texhoma, spoke on Simple First Aid at a recent meeting of the Texhoma Rotary Club.

LEROY A. WOLEVER, M.D., formerly of Midwest City, is now practicing in Shidler.

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PHYSICIANS CASUALTY AND HEALTH ASSOCIATIONS
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Association Represented At Public Relations Institute

Representatives of Oklahoma medical societies recently attended the American Medical Association's 1956 Public Relations Institute in Chicago.

Mr. Don Blair, Associate Executive Secretary of the State Association, Mr. Jack Spears, Executive Secretary of the Tulsa County Medical Society and Mrs. Alma O'Donnell, Executive Secretary of the Oklahoma County Medical Society represented their respective organizations.

The Institute is presented annually as a service to state and county medical societies and is open to representatives of these societies, their auxiliaries, to medical school public relations personnel and to others working in the field of medical public relations.

Three hundred participated in this year's conference, the theme being: "What's Cooking in Medical PR?" Representatives from state and county societies presented public relations "recipes" which had been tried and proven effective in their organizations.

Mr. Blair talked to the group on "The Public Relations Potential of the Medical Detail Man". He outlined the history of the Medical Service Society of America and explained how a close relationship between this organization and the state and county medical societies could benefit organized medicine from a public relations standpoint. Specifically, Mr. Blair enumerated joint projects in Oklahoma where the profession was rewarded as a result of this close cooperation. In summary, he concluded, "Our close relationship in Oklahoma with the detailmen has resulted, in effect, to the addition of some 60 or 70 field representatives to our staff. By virtue of their vocation. these men are considered by many to be spokesmen for medicine—what better group could we find to meet with, work with and educate as to our activities, philosophies and objectives than this made-to-order group of goodwill ambassadors?"

Other discussions covered such topics as: "Local Science Fairs", "Orientation of the Medical Society Newcomer", "Communicating Through Effective Speaking," and "Improving Doctor-Lawyer Relationships."

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Deaths

ISAAC NEWTON KOLB, M.D.
1882-1956

Isaac N. Kolb, M.D., pioneer Blanchard physician died July 31, of a heart ailment.

He was born near Fairy, Texas, July 27, 1882. Doctor Kolb attended medical school at Fort Worth University and received a Doctor of Medicine degree from "Physicians and Surgeons College," St. Louis, Missouri, in 1923.

Doctor Kolb set up his practice in Blanchard in 1924 and has been there for the past fifty years.

JOHN REYNOLDS, SR., M.D.
1874-1956

John Reynolds, Sr., M.D. Muskogee physician died July 14, age 82.

Doctor Reynolds was born in Muskogee, February 26, 1913 and graduated from the University of Arkansas School of Medicine before entering the Army in 1941. After being discharged from the Army he settled in Muskogee.

LEE ROY WILHITE, M.D.
1895-1955

Lee Roy Wilhite, M.D., Perkins physician died July 9, as a result of coronary occlusion.

Doctor Wilhite was born at Helena, Okla., October 17, 1895. He attended the University of Oklahoma and graduated from the University of Oklahoma School of Medicine in 1916.

Doctor Wilhite served three years in the U. S. Army. Following this he located in Perkins, where he practiced until 1942, when he again returned to military service as Colonel, U. S. Army Medical Corps. He returned to Perkins following World War II, and has practiced there ever since.

During his military career Doctor Wilhite was decorated with the Czechloslovakian Military Medal of Merit, the French Croxi de Guerre, the U. S. Legion of Merit, the Bronze Star with Oak Leaf Clusters, and appropriate campaign ribbons.

Doctor Wilhite held offices in county and state associations and served as vice-president of the Oklahoma Academy of General Practice.

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BOOKS RECEIVED

The following books have been received by the *Journal office*. As space permits and the context warrants, books will be reviewed.

CIBA FOUNDATION COLLUQUIA ON ENDROCRINOLOGY. Vol. IX, Internal Secretions of the Pancreas. Editors G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch. and Cecilia M. O'Connor, B.Sc., Medical Book Department, Little, Brown and Company, Boston, Mass. Copyright 1956.

CIBA FOUNDATION SYMPOSIUM ON HISTAMINE. Editors, C. E. W. Wolstenholme, O.B.E. M.A., M.B., B.Ch. and Cecilia M O'Connor, B.Sc., Little, Brown and Company, Boston, Mass., Copyright, 1956.

CLINICAL UROLOGY FOR GENERAL PRACTICE. By Justin J. Cordonnier, M.D., F.A.C.S., Professor of Urology, Washington University School of Medicine, St. Louis, Missouri. The C. V. Mosby Company, St. Louis, Missouri, Copyright, 1956.

DICTIONARY OF DIETETICS. By Rhoda Ellis Ph.D., Instructor of Foods and Nutrition, Department of Home Economics, Brooklyn College, New York. Publisher, Philosophical Library, New York, N.Y., Copyright, 1956.

EPILEPSY AND THE LAW. By Roscoe L. Barrow and Howard D. Fabing, M.D. A Hoeber-Harper Book. Copyright 1956.

HANDBOOK OF PHYSICAL THERAPY. By Robert Shestack, PH.G.R.P., P.T.R. Technical Director, Department of Physical Therapy, Washington County Hospital, Hagerstown, Maryland. Copyright, 1956, Springer Publishing Company, Inc.

IMPARTIAL MEDICAL TESTIMONY. A report by a Special Committee of The Association of the Bar of the City of New York on the Medical Expert Testimony Project. The MacMillan Company. Copyright 1956, New York.

MANAGEMENT OF EMOTIONAL PROBLEMS IN MEDICAL PRACTICE. Edited by Samuel Liebman, M.D., Medical Director, North Shore Health Resort, Winnetka, Illinois: Clinical Assistant Professor of Psychiatry, University of Illinois College of Medicine. Publishers, J. B. Lippincott Company, Philadelphia.

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PHYSICIAN PLACEMENT

The following physicians have expressed a desire to locate in Oklahoma. To the best of our knowledge, the names, addresses, qualifications and availability are current and accurate.

Additional information concerning most of these physicians is available in this office. Anyone interested in contacting these physicians may do so directly or may use the state office as an intermediary.

Anesthesia

Daniel B. Perry, Residence Quarters, Harlem Hospital, New York, N. Y., age 48, Meharry Medical College, 1948, interned at Harlem Hospital, New York and served residency in anesthesia there, veteran, available December, 1957.

General Practice

John R. Lindbergh, Box 217, Weleetka, Oklahoma, Age 29, University of Oklahoma, 1953, Interned in Kansas City General Hospital, Veteran, available immediately.

Internal Medicine

James E. Morris, Jr., 1034 Second St., S.E., Moultrie, Georgia, age 26, University of Tennessee College of Medicine, 1953, one year internal medicine residency, now serving military obligation, available February, 1957.

Pathology

Jess D. Green, Jr., 1765 South Victor, Tulsa, age 32, George Washington University, 1950, will finish four years pathology residency in January, 1957.

Pediatrics

David Goldstein, 66 Lafayette Ave., Staten Island 1, N. Y., age 38, Long Island College of Medicine, 1949, two year residency in pediatrics, Board certified, available after Oct. 1, 1956.

Surgery

Edward Wendell Foster, 147 W. Main, Meriden, Conn., age 62, Harvard University School of Medicine, 1924, certified by specialty board, veteran, available now, would consider surgery, obs.-gyn., or general practice.

Vernon L. Guynn, 2026 S. Second Ave., Maywood, Ill., age 32, University of Illinois, 1947, passed Part I of General Surgery Board, military obligation served, available Jan. 1, 1957.

Boyd M. Saviers, 514 Lacewood Dr., Dallas, Texas, age 33, University of Oklahoma, 1947, finishing third year residency at Methodist Hospital of Dallas, veteran, available September, 1956.

Richard A. Walsh, 250 Fourth St., Elyria, Ohio, age 31, New York University, 1948, completed Part I, American Board of Surgery.

Urology

Harry Emanuel Fisher, Jr., Box 161, Barnes Hospital, St. Louis 5, Missouri, age 33, University of Oklahoma, 1952, veteran, available July 1, 1956.

Henry Ernest Wolfe, Jr., 879 Rosewood Ave., Vallejo, Calif., age 34, Albany Medical College, 1947, board qualified in urology, available upon separation from active duty, Aug. 15, 1956.

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